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CONTENTS

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The British Journal of Tuberculosis and Diseases of the Chest

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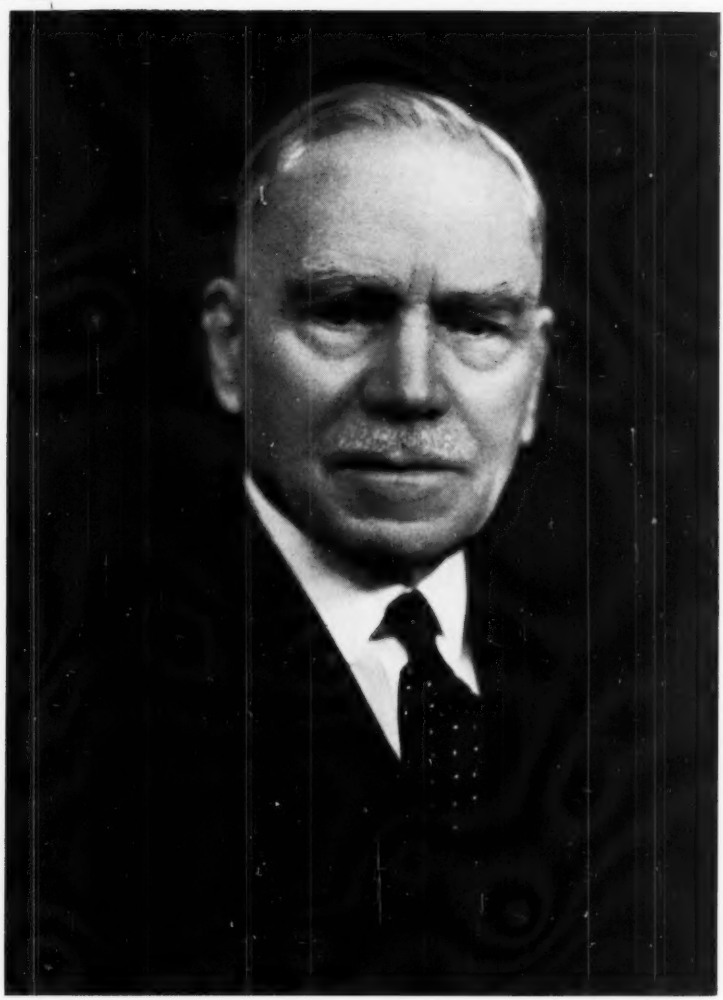

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SIR ROBERT YOUNG

BRITISH JOURNAL OF
TUBERCULOSIS
AND DISEASES OF THE CHEST

Vol. XLVI. January, 1952 No. 1.

EDITORIAL

THIS number of the BRITISH JOURNAL OF TUBERCULOSIS AND DISEASES OF THE CHEST marks the opening of its forty-sixth year, and it is significant of the expansion and importance of the study of diseases of the chest that it has been decided considerably to increase the size of the Journal.

It is with pleasure that we welcome to the Editorial Board four new and distinguished Dominion colleagues:

Dr. Hugh E. Burke, Medical Director of the Royal Edward Laurentian Hospital, Montreal, Canada;

Dr. B. A. Dormer, Chief Tuberculosis Officer for the Union of South Africa;

Dr. Cotter Harvey, Hon. Physician in charge of the Thoracic Unit, Royal Prince Alfred and Royal North Shore Hospitals, Sydney, Australia; and

Professor F. H. Smirk, Professor of Medicine in the University of Otago, New Zealand.

All have kindly consented not only to serve on the Board but also to keep our readers informed, through periodic contributions from experts and others in their respective countries, of the most recent developments arising from socio-clinical research in diseases of the chest, including pulmonary tuberculosis, in their native lands.

In this number Dr. B. A. Dormer inaugurates Dominion contributions with a stimulating and provocative paper on "The Present State of the Chest Services in England and Wales." This is based on a survey made recently by him of the tuberculosis service in this country. His comments on the relationship of the general practitioner to the tuberculosis service may with advantage be compared and contrasted with the informative article by a general practitioner, Dr. G. O. Barber, working in a rural area of England.

We hope that these changes will add to the usefulness and interest of the Journal, both by widening its scope and by drawing into association with us the Dominions overseas.

But this number has an even greater significance, for in it we celebrate the eightieth birthday last November of the doyen of chest medicine, Sir Robert Young, that great physician and teacher, beloved by his innumerable pupils, who has adorned his profession for something like fifty years and whom we are proud to honour. Dr. G. E. Beaumont, his colleague at the Brompton and

Middlesex Hospitals, who has worked closely with him at both hospitals for many years, writes an appreciation of his personality and his work. Sir Robert Young has held almost every high office in our profession. His balanced judgment, combined with a progressive outlook in relation to the vast developments in diseases of the chest, explains the high regard in which he is held by those of us who have been privileged to sit at his feet. We are, therefore, glad to have this opportunity of commemorating his eightieth birthday and of expressing to him our affectionate greetings.

While it is most pleasant to be able to record the progress and activity of the Journal, it should not be forgotten that the burdens of editorship increase correspondingly. We, therefore, take the opportunity of making a heartfelt appeal to our contributors to co-operate with us by reading with care the "Notice to Contributors" which appears in every issue. J. W. Harvie, writing in the *Lancet* this past year, said in his article "On Writing to be Read": "Editors must care for the reputation of their journals, and this care can be exercised with courtesy, reason and discretion without any danger of suppression of individuality." This we wholeheartedly accept as our duty, and we hope that our contributors will bear with us when we attempt to fulfil that duty.

P. E.

SIR ROBERT YOUNG, C.B.E., M.D., F.R.C.P.

"Let us now praise famous men"

By G. E. Beaumont

On November 6, 1951, Sir Robert Young celebrated his eightieth birthday, and this happy event was marked by a dinner given in his honour as a tribute from his colleagues at the Middlesex and Brompton Hospitals.

"R.A.," as he is affectionately known to generations of Middlesex students, has climbed to the pinnacle of success by a combination of genius and hard work. His fame and reputation have become greater with each succeeding year; he has advanced from strength to strength, and now, on his eightieth birthday, he has attained the summit of his career.

I first knew him in 1910 when he was junior assistant physician at Middlesex with charge of the children's department. Later I had the privilege of working first as his house physician and subsequently as his corresponding junior physician at the Middlesex and Brompton Hospitals from 1920 to 1936. When I was a student, R.A. at once impressed me by his meticulously careful clinical examinations, and by his logical and systematic method of diagnosis by a process of elimination. Later, in the wards, his rounds were very popular. He would take a case, enquire carefully into the history, point out the physical signs and indicate the conclusions which could be drawn from them. He would then go over the disease systematically from "ætiology" to "treatment." One of the old students told me recently that R.A. has taught him "bedside manners," for he always treated every patient in his wards with kindness, courtesy and consideration.

Certain of his sayings during the ward rounds linger in my memory. When demonstrating the signs in mitral stenosis, he said, "You may have noticed a firm of carriage builders in Oxford Street called 'Thrupp and Maberly.' The murmur in mitral stenosis sounds like 'Thrupp,'" and he rolled the "r" and accentuated the "p" as he pronounced this onomatopœic word.

When R.A. first went to Brompton the ward windows were kept closed. The physicians eventually decided to keep the windows open, and I remember R.A. telling me how he visited the hospital after the first November fog, in fear and trembling lest he should find his patients acutely ill as the result of the unwonted exposure. At Brompton R.A. demonstrated the importance of first examining the patient and recording the physical signs upon a chest chart before inspecting the X-ray films. R.A.'s post-graduate demonstrations at Brompton were the most popular of any in the course. In these he stressed the importance of a complete examination of each patient from head to foot.

When R.A. retired from active staff of Middlesex his former house physicians presented him with a silver salver on which their names were engraved. The ceremony took place in the board room before a large audience. The senior ex-house physician who made the presentation entertained us by

saying that when he finished his six months as H.P., R.A. enquired what he was going to do. He replied that he was going into general practice. R.A. then said, " I will give you a word of advice." The H.P. waited with eager anticipation for the pearl of great price, for the words of wisdom which would launch him on a successful career. After due pause the sage opened his lips and said, " My boy, always have a good lunch."

During the last war R.A. returned to the wards at Middlesex and delighted crowds of students by demonstrating, in what appeared an almost miraculous manner, how it is possible for the skilled clinician to find out exactly what is the condition of the lungs without first consulting the X-ray films. This art has now, alas ! almost completely disappeared. Those were the days !

There is no space left for me to refer to R.A.'s faithful attendance at the Royal College of Physicians for so many years, or to the numerous honours he received there. Since the introduction of the National Health Service R.A. has done work of the highest value. He was elected chairman of the Board of Governors of the Hospitals for Diseases of the Chest, and by his charm and tact has smoothed away all administrative difficulties and enabled us to pull together as a team.

Very briefly and imperfectly I have outlined some of the outstanding characteristics and abilities of this great physician. As his humble disciple I am honoured at being asked to pay my tribute, and our readers will join with me in wishing him further years of happiness and content in the realisation of work well done.

GENERAL ARTICLES

THE PRESENT STATE OF THE CHEST SERVICES IN ENGLAND AND WALES

By B. A. Dormer

Chief Tuberculosis Officer for the Union of South Africa.
From King George V Hospital, Durban

O, that a man might know
The end of this day's business ere it come!
But it sufficeth that the day will end
And then the end is known.
"Julius Cæsar."

Early attempts at the control of tuberculosis, which from the time of Koch's discovery of the tubercle bacillus was regarded as an infectious disease, largely neglected the paramount importance of environmental factors and was responsible for the first separation of tuberculosis from the general stream of medicine. Hence the advent of sanatoria, which created an isolated home for cases of tuberculosis.

These sanatoria, some large, some small, in the beginning treated the disease by means of rest and climate, and much later used collapse therapy of various kinds, both minor and major, even the somewhat primitive major surgery being done by physicians. Sanatoria meant isolation for the patients and mental as well as physical isolation of the medical, nursing and other staff. Sanatoria vied with each other in producing amenities for their patients and staff in order to keep them happy in their remote surroundings, and eventually a great complex of buildings arose around the parent institution giving an enormous façade to a comparatively few patient beds. Sanatoria were owned by large Local Authorities, by combinations of small ones, or were private institutions.

The next step in the separation of tuberculosis from general medicine was the concept of Sir Robert Philip—the tuberculosis dispensary with its doctors, which was the focal point of a tuberculous patient's life. This led to the evolution of a tuberculosis officer, whose sole duty it was to care for the tuberculous, mostly in the dispensary, but to a certain degree in their homes by what was called "home visiting." His contact with pulmonary disease apart from tuberculosis was extremely limited and his association with general medicine was remote. He worked under the medical officer of health of a Local Authority which was responsible for all aspects of the care of all forms of tuberculosis, including that of the bones and joints, the genito-urinary system, the skin, the lymphatic glands, etc. Prevention, diagnosis, treatment in a minor degree (as all cases discovered were supposed to go to sanatoria, apart from certain advanced cases who were accommodated in hospitals for

infectious diseases), and after-care, were all functions of the tuberculosis officer. He had also certain statutory duties, such as the keeping of a tuberculosis register of notified cases, a function delegated to him by the medical officer of health.

With few magnificent exceptions local authorities did not provide the tuberculosis officer with suitable quarters, and dispensaries were usually ramshackle affairs established in dwelling houses and without the proper tools for skilful diagnosis (X-ray sets, etc.). The average Local Authority did not insist on a high quality of officer, nor did they offer salaries to attract the brilliant man.

Three types of tuberculosis officer emerged: the man who used the tuberculosis dispensary as a part of the training of a medical officer of health and who had no abiding interest in pulmonary disease; the man who had had tuberculosis himself and was interested in the disease in a missionary way; and, lastly, the man who just drifted into a tuberculosis appointment because he could get nothing better.

A malign result of the removal of tuberculosis from general medicine was that this disease was badly taught. It became the habit of most teaching and general hospitals and of general practitioners to get rid—to the tuberculosis officer—of a case of pulmonary tuberculosis as soon as it was diagnosed.

Such was the background during and after the first World War when, some years before the National Health Service Act came into being, general surgeons began to take an interest in thoracic work and some of them became primarily thoracic surgeons, their interest being not solely in non-tuberculous conditions, but extending to tuberculosis itself. It was soon discovered that existing sanatoria were not suitable for this rapidly expanding form of treatment, and feverish attempts were made to construct new institutions suitable for thoracic surgery and to establish operating suites in at least some of the larger sanatoria. It was this rise of thoracic surgery which stimulated new interest in tuberculosis, which had been jogging along in a somewhat complacent way for years.

The second World War and the post-war period brought tuberculosis into much clearer focus. The great exodus of the tuberculous from their sanatoria to make room for bomb casualties, the expected epidemic of tuberculosis which did not occur, roused the curiosity of more than the epidemiologist, and some people began to wonder if, after all, the right course had been taken when the final and complete separation of tuberculosis from medicine took place.

Waksman's discovery of streptomycin and the use of chemical substances such as para-amino-salicylic acid gave the tuberculosis world another severe jolt, and it is noteworthy that the modern approach to the treatment of pulmonary tuberculosis came from without and did not emanate from the tuberculosis fold.

When the National Health Act came into being it created a chest physician and brought the wide spectrum of pulmonary disease, including pulmonary tuberculosis, under his care, and made salaries of consultants sufficient to attract young men of quality. It brought pulmonary tuberculosis, as a disease for hospital and specialist diagnosis and treatment, under the Regional

Hospital Boards and placed prevention and after-care under the Local Authority, but implied that the chest physician would be responsible for part at least of these Local Authority duties. Thus it was implied that the chest physician was different from other consultants as, if he performed the duties of tuberculosis officer of the Local Authority, he would do domiciliary visiting (as contrasted with a consultant visiting a patient's home with his general practitioner), take charge of the tuberculosis register, do contact examinations, and give B.C.G., none of which functions were accepted as the duty of any other consultant.

It appears that the question of non-specialist diagnosis and treatment was left to the general practitioner. However, a comparatively new technique of diagnosis, mass radiography, was to X-ray large numbers of healthy-appearing persons and thus provide the early asymptomatic cases of tuberculosis and of other thoracic abnormalities—significant or otherwise. This was to work under the Regional Boards, but to derive technical guidance from the Ministry of Health's Centre in Tavistock Square.

At this stage it is necessary to discuss the question of the functions of a general practitioner, chest consultant, and medical officer of health to a Local Authority in order to obtain a clear picture of the present status of pulmonary disease in relation to the National Health Act.

1. *Regional Boards Chest Consultant.*—A consultant is usually an individual who applies specialist knowledge and techniques for the diagnosis of cases referred by a general practitioner. Should the treatment of such a diagnosed case be within the capacity of a general practitioner he returns the case to him with advice. If the treatment requires specialist knowledge and techniques, he will treat the case in one of his beds in hospital, ultimately referring it back to the general practitioner, but it may be necessary for the patient to return periodically to the specialist for treatment or survey. On occasion it will be necessary for a consultant to see a patient in consultation with the general practitioner in his own home. In the face of an emergency such as lack of beds in hospital, it may be necessary for a consultant to use specialist techniques, in co-operation with a general practitioner, in a patient's own home.

2. *General Practitioner.*—A general practitioner is usually considered to be a "family" doctor, whose intimate knowledge and contact with the individual leads him to become readily aware of any disturbance of normal physiological function in any of his patients. He is expected to diagnose the cause of such disturbances, if they come within his techniques and experience, and to treat them if they come within the same spheres. Should the diagnosis or treatment be beyond him he refers the case to a consultant—in the case of pulmonary dysfunction, to a chest physician. One would expect him to contact the medical officer of health of his Local Authority should environmental conditions be suspect as a cause of his patient's condition or hinder his recovery. He would also, under present conditions, call in the aid of the medical officer of health should his patient need home help, health visitors or home nursing, or an ambulance if he has to go to hospital.

3. *A Medical Officer of Health* of a Local Authority has numerous important functions with regard to the community. He must see that it is provided with

proper housing, clean and pure food, milk and water, and adequate sewage disposal. Other important duties include the prevention and control of infectious diseases, the health of school children, maternity and child welfare, after-care of tuberculosis, the provision of home helps, health visitors and home nurses, and the provision of transport for patients needing diagnosis or treatment away from their homes. He is responsible for the education of the public in health matters. In the discharge of these duties he may use the services of others, such as school medical officers for tuberculosis testing and B.C.G. vaccination of school children, of general practitioners for the protection of young children against diphtheria or for the giving of B.C.G. to new-born infants.

In this view of the functions of the various persons concerned with the care of the individual's health I find it difficult to envisage the rôle of a chest consultant as a tuberculosis officer of a Local Authority. After careful reading of the Ministry's booklet on the development of consultant services I find little justification for regarding a chest consultant, as compared with a general physician or a consultant in infectious disease, as a person who keeps (for an M.O.H.) the tuberculosis register under the Tuberculosis Regulations of 1930; who sees non-pulmonary tuberculosis (bone, joints, genito-urinary, glandular, skin, meninges, etc.) and who is, like the old tuberculosis officer, a specialist in such subjects; who goes to the patient's home in the absence of the general practitioner (performs or fulfils the rôle of a general practitioner); who receives reports from health visitors; who collects contacts of cases of tuberculosis—who, in fact, contains in one individual the functions of a chest specialist, a tuberculosis officer in charge of all forms of tuberculosis, and a general practitioner in cases of tuberculosis. One important point is obvious—a consultant in infectious diseases is not expected to gather the contacts or immunise the susceptible, yet the chest consultant is expected to do both.

The New Outlook

What has happened to tuberculosis since the appointed day, and how are pulmonary diseases faring?

A. The Regional Board.

Functions.—Hospitals and their staff. Consultant services, including chest physicians and thoracic surgeons. B.C.G. vaccination of nurses and students. Mass radiography.

It must be said at the outset that the appointment of chest physicians under a Regional Board has improved conditions in this sector of medicine beyond recognition. In each Region visited there has been incredible improvement in the provision of Chest Clinics—the old tumbledown tuberculosis dispensary (converted dwelling house) with no tools for diagnosis or treatment is everywhere giving way to Chest Clinics situated in, or as near as possible to, general hospitals—the ultimate aim being to have every Chest Clinic an integral part of a Medical Centre (General Hospital and Infectious Diseases and Chest Departments including pulmonary tuberculosis). X-ray sets have been installed or are projected and other necessary instruments of diagnosis and

treatment have been provided or are scheduled. In particular, in those regions where formerly an apathetic Local Authority held sway is the improvement most obvious.

Chest Physicians

A large number of enthusiastic chest physicians of quality have been appointed, and their effect is already being demonstrated by a considerable increase in patient attendances at Chest Clinics. In many instances attendances have trebled. This fact alone has accounted for a considerable increase of notifications of tuberculosis.

With regard to the rôle of the chest consultant *vis-à-vis* the Local Authority, there have been several interpretations of the National Health Act. One is that the chest physician regards himself as an adviser in tuberculosis to the Local Authority and is prepared to give advice when asked; another is that the chest physician is the tuberculosis officer of the Local Authority—this is the view held by the older type, those who graduated from tuberculosis officer to chest consultant; and there is the third view of the "new type"—that the chest consultant is a specialist in general medicine with a leaning to pulmonary disease and is no different in functions from any other consultant.

With regard to remuneration, some Regional Boards pay the chest specialist's whole salary and hope to collect $\frac{2}{11}$ths from the Local Authorities. Other chest physicians have two contracts—one with the Regional Board for $\frac{8}{11}$ths and one with the Local Authorities for $\frac{3}{11}$ths of their salary, but BOTH at full consultant rates. Others have similar dual contracts, but the Local Authorities' $\frac{3}{11}$ths is paid at administrative rates. Others still have no contracts with the Local Authorities yet, and work for their Regional Board at $\frac{8}{11}$ths of their normal salary. One feels that a clear definition of function is necessary, as the majority of chest consultants are not happy over this present apparent muddle. It has been indicated in a review of function that it is not really logical to assign different basic duties to one sort of consultant—the chest physician. The relationship of chest physicians to medical officers of health appears to be lacking in harmony because of these differences of outlook. It must be remembered that the training of the true chest consultant—*i.e.*, general medicine in relation to pulmonary disease—no longer includes a period as an assistant medical officer of health or as a tuberculosis officer, and that therefore his concept of his duties is not that of an administrator who has been trained in public health. The medical officer of health often feels that tuberculosis has been unfairly taken away from him and given to a youngster who, as a graded consultant, gets much more pay (and is therefore considered more important) than himself.

The chest physician, because of this, often fails to see why he should bother with a tuberculosis register or domiciliary visiting or attendance at care committees or housing committees, when his time is taken up with the greatly increased attendance of patients for general pulmonary diagnosis and treatment. It appears obvious that the better the chest physician the greater the attendance of patients referred by general practitioners, and the less time he will have to do more than fulfil his functions as a consultant.

Relationship of Chest Consultants to Other Consultants except Thoracic Surgeons

A certain number of general physicians interviewed, especially those interested in chest disease, were very suspicious of the chest physicians and considered that they were merely tuberculosis officers disguised as consultants. Others were co-operating admirably with their new colleagues. Children's specialists were anxious to take over the young child with tuberculosis, but were prepared to accept their new colleagues as part of the team. Orthopædic surgeons felt that bone and joint tuberculosis was part of their job, as did skin specialists with regard to lupus. Radiologists and chest physicians are still finding their relationship a little strained. In the past the sanatorium physicians and some tuberculosis officers have been accustomed to use X-ray apparatus in comparative isolation and have developed ideas and techniques independent of the radiologist, who feels that the whole subject of radiology is his. Compromise is being achieved on the lines that the radiologist is technically in charge of X-ray apparatus and can delegate parts of this to the chest physician, using himself as an adviser on difficult techniques and as a consultant when interpretation of difficult plates is necessary. An important point is that radiological services, particularly X-ray of the thorax, should be available in increasing degree to the general practitioner without unnecessary barriers.

When the chest physician is officially on the staff of the hospitals in his area and proves his qualities, all these teething difficulties will disappear.

Beds

Most of the teaching hospitals were not interested in the provision of beds for tuberculous patients, believing that tuberculosis cases ought to go elsewhere. One feels that until the teaching hospitals provide beds for tuberculous cases and some sort of thoracic surgery department in their own sphere students will not be well taught in thoracic disease. A study of the records of cases of carcinoma of the bronchus, bronchiectasis, asthma, emphysema and bronchitis in the teaching hospitals I visited showed the disadvantage of not having a thoracic team (physician and surgeon) associated with the teaching hospital. One possible solution from the point of view of teaching would be the appointment of a chest physician and a chest surgeon jointly by the teaching hospital and the Regional Board. This would give a stimulus to the teaching of pulmonary disease, which is commonly regarded as an extra academic activity and not part of hospital life.

General Hospital Beds

It is extremely difficult to divert beds in general hospitals to pulmonary tuberculosis, owing to great pressure on them and the rooted objection of general hospital staff to change of any kind, especially the introduction of an infectious disease into a general hospital. One of the greatest possible helps in this direction would be to utilise the idea of the 800-bedded hospital centre with its general, medical and surgical beds, its infectious diseases wing, its chest division including pulmonary tuberculosis, immediately. Instead of waiting until these compact medical centres are actually built, would it not

be better to regard the local general hospital, the local infectious diseases hospital, the local sanatorium, however distant they be from each other, as a hospital centre, the infectious diseases and tuberculosis divisions being regarded as pseudopodia? Such a centre would have one staff, one matron, for example, with assistants on the periphery, and would be known as one centre. Nurses would automatically be taught in all hospitals of the centre and would not think of objecting to nursing cases of tuberculosis or of infectious disease as this would be part of their normal duties.

Beds in Infectious Diseases Hospitals

Most Regions are deliberately utilising every possible bed in infectious diseases hospitals, and these will form a good beginning for the education of both medical and nursing staff that looking after tuberculosis is part of everyday hospital life. In some cases wards in infectious diseases hospitals have been turned into chest clinics, but this is and should be regarded as a temporary measure or as a part of the out-patient department of a hospital centre.

Sanatorium Beds

These offer singular difficulties in integration, as most sanatoria are isolated and have grown up under the medical superintendent, who, as senior physician to the institution, was in the past the most important personage in the tuberculosis world. He received his cases from his colleague, the tuberculosis officer, and it was considered essential that every tuberculous patient should, for some period of his life, reside at a sanatorium for (1) education in a way of life, and (2) treatment. This led to the patient having divided loyalties to general practitioner, tuberculosis officer and sanatorium physician, the last usually having the most influence. Most sanatoria today are not suitable for thoracic surgery, but some have been adapted and one or two could be used solely for surgical procedures. In any case, the utmost use could probably be obtained by utilizing sanatorium beds as part of a hospital centre and by placing the actual beds under one or more chest physicians' or thoracic surgeons' direct care.

In some Regions chest physicians had both tuberculosis and non-tuberculosis beds at the local general hospital, tuberculosis wards in the local infectious diseases hospital, and some beds in the local sanatorium. This is the ideal and could easily be carried out by all Regions. A chest physician without beds is a tuberculosis officer of the old school under another name, and will achieve nothing. Even if beds are not immediately available the chest physician should most certainly be officially on the staff of the general hospitals in his area and should have token beds. This has been done by some Regions and could be universal.

Children's Sanatoria and Children in Sanatoria

One was struck by the large number of healthy-looking children with primary tuberculosis running about or at school in ordinary sanatoria and in special children's sanatoria, and one feels that beds occupied in this manner would be better filled with young adults or others requiring active treatment.

There are empty beds in every Region, the reason being lack of nursing staff, for Britain is no exception to the world-wide shortage of nurses. This is felt most keenly in the sphere of pulmonary tuberculosis, owing to its being regarded as one of the least attractive types of nursing. An interesting experiment would be the setting up of nursing colleges in association with technical colleges or universities, which colleges would supply groups of nurses to the hospital centre for training.

Some Regions have a central bed bureau; one in particular had a large section in a building of its own, devoted to this purpose; but such machinery is cumbersome and costly. The chest physician with his own beds is in the best position to judge who should occupy them and for how long; the only justification for a central bureau is where it finds beds when the chest physician has filled his own and needs an overflow.

Decentralisation rather than centralisation, with intimate contact between chest physician and patient and closeness of the patient to his home and family, seems to be the real solution.

Domiciliary Treatment

This term has come to mean, at the moment, specialist domiciliary treatment—*i.e.*, artificial pneumothorax, pneumoperitoneum, etc. In view of the acute shortage of beds for the active treatment of pulmonary tuberculosis some chest physicians have been in the habit of treating cases of pulmonary tuberculosis at their homes, using specialist techniques normally available only in hospitals. This bed at home is really an extension or pseudopodium of a hospital bed to the patient's home and should be regarded as such. Much credit is due to those chest physicians who, not content to wait while beds for specialist treatment were available, decided to carry out treatment in the patient's home. As a practical contribution to the present problem of " beds for the tuberculous " it is of the utmost value and should be pushed to its limit in every Region. Its proponents feel that at least 70 per cent. of newly diagnosed cases can be so treated and cured without at any time occupying a bed in hospital. The enthusiastic co-operation of the medical officer of health of a Local Authority can often do much to make such treatment possible in homes in crowded areas.

Non-Tuberculous Pulmonary Disease

A great increase in the efficiency of the diagnosis and treatment of non-pulmonary conditions has taken place since the appointed day. Bronchiectasis, asthma, emphysema and carcinoma of the bronchus are all being seen in greater numbers by Chest physicians and diagnosed and in some cases treated earlier than ever before in this country. This service is growing rapidly and will continue to grow. The comparative rarities, such as Boeck's sarcoid, are receiving attention, as are the supposedly, yet not truly, rare industrial chest diseases. In these non-tuberculous diseases alone the appointment of chest physicians has been amply justified.

Thoracic Surgery

This is the most difficult sector of chest disease to view clearly. Thoracic surgeons in Great Britain grew up in splendid isolation, and being very few in number became most precious to the community. The war gave such an impetus to thoracic surgery that today, when it forms a most important part in the treatment of many thoracic conditions both of the heart and lungs, these surgeons cannot manage the volume of work which awaits them. In some areas patients have to wait up to two years for a major thoracic operation, and this particular point is one of the few real issues in the causation of the so-called " crisis in tuberculosis." Chest physicians feel up against a wall of frustration when their cases are in need of surgery. This is almost general; a very few areas have been lucky enough to be able to arrange surgical treatment fairly quickly. There are many reasons for this waiting list:

1. Lack of suitable beds. This is mostly a need for operating theatres, and every effort should be made to provide operating facilities in any hospital where thoracic surgery can be done.
2. Lack of nursing staff. This will eventually be overcome, but more rapidly if nurses are trained in Medical Centres of which the tuberculosis beds are a part. It is no good directing or seconding nurses from teaching hospitals. This also perpetuates the psychological error of regarding nursing cases of tuberculosis as dangerous and not part of ordinary duties.
3. Lack of sufficient surgeons. This can easily be overcome by training and appointing more surgeons.
4. The fact that the chest physicians are finding more and more cases suitable for thoracic surgery.
5. The fact that the use of antibiotics and chemotherapeutic substances renders many more cases of tuberculosis suitable for surgery which in the past would have died.

Nobody can be blamed for this situation, but one or two points should be made. The association between chest physician and thoracic surgeon is seldom close enough. In fact, in some areas they conduct parallel clinics in competition. The correct procedure is obvious—that a thoracic surgeon and his physician colleague should work as a team. As far as can be ascertained the average thoracic surgeon can do the surgical work for two chest physicians, and it was also discovered that the most efficient work seemed to be done by those surgeons who worked with a physician in a sub-Region as an independent team and not attached to some regional headquarters. If newly diagnosed cases of pulmonary tuberculosis were seen by the chest physician, the thoracic surgeon and the general practitioner in consultation, the future of the tuberculous patient would be bright. The same holds for other pulmonary diseases requiring surgery. Regional Boards seem to be well aware of the difficulties facing them with regard to thoracic surgery and are doing all they can to solve them. The utmost use of beds so that those that are available are used for active treatment, more willingness on the part of thoracic surgeons to operate in theatres which are not palatial but which are functionally usable,

will help. In some areas, where the greater proportion of surgical beds are devoted to non-tuberculous conditions such as advanced carcinoma of the œsophagus or bronchus, it may help considerably if more such beds are devoted temporarily to the treatment of young adult cases of tuberculosis with a good prognosis. Some consultants suggested that the best thing to do would be to send cases requiring major surgery to other countries which have beds available—*i.e.*, Switzerland. This would lead to incredible complications and in the end would defeat its own object.

Mass Radiography

The purpose of mass radiography is to X-ray large sections of or even the whole of the population with a view to finding, amongst the apparently healthy, early asymptomatic cases of tuberculosis, cases of other thoracic abnormalities, and cardiac lesions both congenital and acquired. In all the areas visited mass X-ray teams existed, the average team of workers being eleven to each set. Most were housed in buildings of their own, and in one or two areas there existed a Central Mass X-ray Department which catered for as many as five teams. This elaborate system exists to discover comparatively few cases of tuberculosis each year. Over 95 per cent. of the notifications of pulmonary tuberculosis originate in general practitioners' surgeries, and if the miniature X-ray apparatus were at the disposal of patients visiting general practioners because of some disturbance of normal physiological function and for the use of contacts of cases so discovered, much greater value would be obtained. In some areas there was a tendency for the mass X-ray team to run a parallel chest service, but most Regional Authorities felt that a closer approximation to the chest physician was indicated, and actually had the mass X-ray director do sessions at the Chest Clinic.

A mass X-ray apparatus is a piece of diagnostic machinery, and should be put at the service of a chest physician to X-ray such sections of the community in which he is most likely to find tuberculosis, remembering that it is the general practitioner's right to see patients first. Miniature X-ray attachments costing comparatively little could easily be fitted to existing X-ray plants in Chest Clinics, and the general practitioners provided with a round-the-clock chest X-ray service.

Use of B.C.G. in Regional Hospitals Nursing Staff and in Teaching Hospital Staff and Students

Most teaching hospitals are not yet sufficiently educated in prophylaxis of disease in their students or staff. However, in the comparatively short space of twenty months considerable progress has been made and the pace is increasing. Thus, up to the end of May 1951 sufficient vaccine had been applied for and issued to vaccinate more than 16,000 hospital nurses and medical students. The Medical Officer of Health should be regarded as the central figure of the whole scheme in his area, and the use of B.C.G. is really a delegated one.

B. The Local Authority: Services

Functions.—With regard to tuberculosis and chest disease, prevention, including the use of B.C.G. vaccine, night sanatoria, hostels, etc. Receipt of notifications of tuberculosis, keeping the tuberculosis register in terms of the Tuberculosis Regulations of 1930, housing of the tuberculous, contact examination, after-care, almoners, maintenance of provision of home nursing services, home help, health visitors, ambulance services, transport of patients for treatment, etc.

Nearly all the medical officers of health interviewed were disgruntled and felt that tuberculosis had been snatched from them and handed over to the Regional Boards. They felt acutely the loss of their hospitals, sanatoria, infectious diseases wards, their tuberculosis dispensaries, their tuberculosis officers.

Most of them felt that the $\frac{8}{11}$ths or $\frac{3}{11}$ths which the Local Authority did or did not pay of the chest physician's salary represented their full responsibility to tuberculosis, and further expected the chest physician to carry on in the same way as the old tuberculosis officer. Their colleagues of the Town Clerk's Department and the City Treasury felt that, as tuberculosis was now an affair of the Regional Boards, they could rest content on a minimum of expenditure.

Some medical officers of health regretted the fact that since the tuberculosis officer had become the chest physician and had moved his clinic to the general hospital they no longer had personal contact with him. Some of them were frustrating the chest physician by holding up or refusing to obtain for him B.C.G. forms, as they had up to date no formal direction as to the duties of a chest physician. Others impeded his work in various ways according to how they defined their function.

Tuberculosis is a disease of environmental stress, and it is the duty of the medical officer of health to see that environmental conditions are suitable for the patient on his return from hospital.

A good medical officer of health can help the adolescent factory girl and the male manual worker to understand the causes and risks of tuberculosis, and assist them to avoid too much fatigue and other stresses where they are most dangerous. He can try to protect the right people by means of B.C.G. vaccination of infants, school leavers on whom environmental stress will soon be pressing, contacts of cases of tuberculosis. The primary responsibility rests with him and not with others. Most of the medical officers of health interviewed left propaganda and education in chest diseases, including tuberculosis, to health visitors and to chest physicians. When it is realised that most tuberculosis patients spend the greatest part of their lives at home with their families and, if they have their own way, all of it at home, the importance of educating the patient how not to spread disease is paramount. Propaganda should be oriented in terms of modern psychology and the patients turned into militant co-operators in the anti-tuberculosis campaign. Once patients realise that they need not be infectious if they put an "invisible barrier" round themselves by following simple procedures, and that by so doing they are actively aiding in combating tuberculosis, the majority of them co-operate

enthusiastically. It is one of the functions and indeed the inescapable duty of the medical officer of health to educate the public and the patients properly, insistently and without relaxation.

One does not feel that the provision of night sanatoria or hostels for the chronic case of tuberculosis will be of much avail. Out of some sixty chronic cases interviewed only two stated their willingness to go to such places, preferring their homes and families to any form of institution.

The training and payment of relatives of the patient as a home nurse or help is one of the many ways a medical officer of health could use to care for the patient in his home, and the use of such trained persons to take in chronic cases as lodgers when the house contains adults would solve the problem of the homeless case.

Rehabilitation of the tuberculous is one of the most difficult problems known to man; but the medical officer of health and the general practitioner between them, with the help of the National Assistance Board, the Disabled Persons Act and Care Committees, will in general be able to help the case needing such rehabilitation. With earlier diagnosis, the application of chemotherapy and antibiotics to treatment, even the use of chemical substances for prophylaxis, and definitive thoracic surgery at the right moment, there should be fewer and fewer cases requiring rehabilitation. The emergence of a notified case of tuberculosis in such a highly organised society as in Britain means the failure of prevention and ultimately a failure on the part of the environmental specialist, the medical officer of health. It is an extraordinary thing that the medical officer of health is fully aware of his responsibility in preventing diphtheria, smallpox, whooping cough and measles, but now often feels that tuberculosis belongs to somebody else, the somebody being the Regional Hospital Board's chest physician, who does the medical officer of health's share of the work for $\frac{5}{11}$ths of his salary.

C. The General Practitioner

Functions—He detects disturbance of normal physiological functions in his patients, he treats within his capabilities, and he refers his patients to consultants when he requires specialist diagnosis or techniques in treatment.

The average general practitioner interviewed thought tuberculosis a responsibility of the Regional Board since they took it off the Local Authority. He felt that today it took much longer to " get rid " of cases of tuberculosis to institutions. " Get rid of " are the operative words, and reflect his teaching and the general attitude to tuberculosis. He still believes that the case of tuberculosis should rest in bed until admitted to an institution for treatment. He admitted that the quality of service rendered by the chest physician was much better than in the past, and he was sending more and more cases of all types of chest disease (asthma, bronchitis, emphysema, bronchiectasis, etc.) to the chest physician. He admitted that he was so pushed today with so many more attendances at his surgery, so many more visits to do, and inadequate remuneration, that he tended to shift patients off to anybody who would see them.

He did *not* realise that over 95 per cent. of all cases of tuberculosis notified

came from his surgery, that most of them were diagnosed too late, and that at any one time over 50 per cent. of the positive spitters on a dispensary register were under his care. Now that antibiotics and chemotherapeutics substances are easily obtainable the general practitioner will take a greater part in the treatment of tuberculosis than ever before. Some general practitioners of better quality were already taking a great interest in the newer forms of treatment and were either working with the chest consultant or treating the patients on their own.

It was interesting to see in each Region visited the complex arrangements that existed to provide the patients with these chemotherapeutic and other special drugs. In some areas the consultant kept a stock of his own in the same way as the tuberculosis officer used to keep malt and oil. Other chest physicians prescribed the drugs, and still others wrote to the patients' general practitioner asking him to prescribe. The last appears to be the only correct procedure.

One feels that the general practitioner ought to be educated in his present great responsibility for the care of cases of tuberculosis. With early diagnosis—and this will come with education and the provision of better radiological facilities—and with advice from the chest physician, he will be able to treat many cases at home. He has the help of the medical officer of health in the shape of home helps, home nursing services to give injections of antibiotics, health visitors and almoners, and the medical officer of health to adjust housing, work and other environmental conditions out of gear.

D. The Patient

What is the viewpoint of the man in the street—the ordinary citizen, the subject of all this bother? As far as could be judged, he did not think about it at all unless he felt ill. He then went to see his doctor, and although he admits he had no anxiety about the bills, he felt he had to wait longer and got less attention.

He also felt he had to wait longer at out-patient departments both for consultations and X-ray, and he had to wait longer for a bed in a sanatorium. Only a comparatively few dodged the waiting by going privately to a doctor or specialist.

He realised that a lot more people were seeking medical advice today, but said there ought to be more doctors, " so that you didn't have to wait so long."

He still looked on his doctor as the important part of his medical life, but felt a bit bewildered by different loyalties to doctor, to chest consultant, to sanatorium physician. He preferred home to hospital, but when in hospital usually submitted cheerfully to anything the " docs " thought best for him. He wanted to get home as soon as possible and liked to see visitors when he was in hospital.

He preferred to go back to proper work than be rehabilitated. He thought night sanatoria and hostels were " daft."

He did not like mass X-ray teams coming to factories, " as the other chap suspected you had T.B. if you were off work if they called you up twice."

He sometimes cheated the N.H.A. in various ways; but this one was a *rara avis*.

To sum up my impression: there is no doubt in my mind that the provision of subsidised foodstuffs and the institution of rationing in Britain, so that the poorest can obtain sufficient food of adequate quality, superimposed on a firm basis of good environmental conditions (housing, pure water, adequate sewage), will in the end, by the removal of environmental stresses which render man a " susceptible animal " (in terms of Koch's postulates), prove the greatest and most important factors in the eventual control of tuberculosis in this country.

The National Health Service is providing skilled diagnosis and treatment through its Regional Boards and chest physicians.

Certain teething difficulties are being experienced.

If the Local Health Authorities take their proper place in prevention, if there are more practitioners and they are better educated in thoracic disease and provided with more radiological facilities, there will result a spectacular drop in the death rate and incidence of this disease. In my opinion, the " T.B. Crisis " so publicised in the press was a temporary check in the great and successful offensive against tuberculosis in Great Britain.

The author has worked in general practice, in teaching hospitals, in sanatoria and Local Authority Health Departments in Great Britain.

The following types of person were interviewed to give material for this article, the interviews taking place during 1946, 1948 and 1950:

1. Senior officials of the Ministry of Health.
2. Senior Administrative Medical Officers of Regional Boards and their staffs, including chest physicians and sanatorium physicians and thoracic surgeons, matrons, nurses.
3. Medical officers of health.
4. Town clerks and city treasurers and town councillors.
5. General practitioners.
6. Chemists.
7. Tuberculous patients.
8. Teaching hospital staffs, including doctors, matrons, nurses.
9. Senior medical students.

THE PLACE OF THE GENERAL PRACTITIONER IN THE TUBERCULOSIS SERVICE

By G. O. Barber

From General Practice at Great Dunmow, Essex

His Intimate Knowledge of his Patients

"Captain of the Men of Death." In Osler's day the claim of tuberculosis to this title was unchallenged. Even twenty-five years ago there was still little that medicine could do except to delay the course of the disease, and the general practitioner had to watch his patients' deadly conflict, powerless to help save by promoting the most favourable conditions for body and mind to put out their whole resources against the invading bacillus.

I remember asking my chief, "But what treatment is he having?" and his reply, which left me unsatisfied, "Bed, fresh air, and reassurance."

Today the position is greatly altered, and I am constantly astonished at the apparent recovery of my tuberculous patients, especially if they are sent *early* to the chest physician. To illustrate this the following are the figures in this country practice. Out of about 3,500 patients on the doctor's list, there are 30 patients affected. These patients are in the following stages:

Under investigation but not yet finally discharged	4
Diagnosed and awaiting admission	0
Under treatment in hospital	2
Successfully treated in hospital, and attending out-patients	10
Discharged and apparently cured	14

(Not including adults who suffered from adenitis in childhood.)

During the past year one patient has died of tuberculosis.

On paper these are just figures. But to the general practitioner each unit is not only a well-known individual, but a member of his particular little flock of families, families with whom he has lived in close association for all of his professional life, and on whom he has been practising what is now called "social medicine." He has not only seen them for occasional illness, but he has been intimately concerned with every phase of their lives, and with all that conduces to health or disease.

Social medicine teaches that the family rather than the individual is the unit. The general practitioner has always done this especially in relation to tuberculosis, and for this reason he can be more accurately referred to as a *family doctor*.

Indeed, it is in the personal attitude that the family doctor differs from the consulting chest physician. The latter sees more of the *disease*, whereas the general practitioner has the wider picture of the *individual* patient and his family and his environment. In the special department of the hospital, and even in the chest clinic, the cases have already been sorted, so that they present themselves for the first examination either as a definite case of tuberculosis or as one which is strongly suspected; they are guilty until they can

be proved innocent; and they tend to be well-established cases. That is the stage at which they have left the hands of the general practitioner. It is this preliminary pre-hospital period with which the general practitioner has to deal, and where his place is of the first importance in the whole tuberculoss service.

Early Diagnosis

Because the chances of survival of the patient still depend largely on how early the diagnosis is suggested, the family doctor's main duty is to distinguish the first insidious symptoms of a deadly disease out of the great mass of minor illness with which it is his main livelihood to deal. It has been my own experience in twenty-one years of general practice that this " spotting " of the early stages of tuberculosis is something which is only acquired gradually and painfully, for nothing can cause the conscientious doctor greater pain than the realisation that he might have made his diagnosis sooner. One reason why the young doctor finds this difficult lies of course in the very nature of his training. During his time in hospital he has looked at tuberculosis from the point of view of the consultant, for he has been taught only on the well-established cases.

Early Cases

He does not meet the early cases until he leaves hospital and starts to be the first contact with medicine for the little group of the community which he calls his " practice." These people are in his care, they consult him for every little breakdown in the normal working of their bodies, and it is his duty to deal with them to the utmost extent of his ability, knowledge and resources. Of course he does not inherit from his predecessor a set of completely healthy human beings, for when he takes over a practice it includes a number of ill patients, and I well remember the half-dozen advanced cases of pulmonary tuberculosis who lay coughing out their last days when I first was introduced to them. It was some time before I realised to my horror that *new cases* were occurring around me, insidious, with no red flag hoisted to warn themselves, or me, that they were in fact already marked men and women who would in time replace these other advanced cases as they finally died one by one. It is only when the young doctor realises this gruesome truth that he begins to be successful in the detection of early tuberculosis.

Once he achieves this awareness, he should be ready to assume the responsibility of his position in the general tuberculosis service. And so I repeat that by far his most important duty is the detection of the disease in its earliest stage, so that he may place it rapidly under expert care.

He will probably lose sight of his patient for quite a long time but if treatment is successful then much later he has the aftercare of the convalescent patient on discharge from the hospital.

Let us then look at this duty of early diagnosis in more detail. I remember being taught, when considering any disease, to ask myself a routine question, " Could this be due to syphilis or to tuberculosis ?" and I believe this to be sound teaching. It does not mean to say that every case must be subjected to every routine laboratory test. But it does mean that the general practitioner ought to be much more aware of the early symptoms of the disease. If he

waits for all the classical signs which he learns in hospital, he is going to waste months of valuable time. He has to learn, sometimes by sad experience, that the early manifestations are small symptoms, and it is the teaching of these which ought to be emphasised during his student days.

To deal first with pulmonary tuberculosis. What is it that first brings the patient to the doctor? Cough?—sometimes, but so much more often it is just a *lack of well-being* without any obvious cause, which should make the doctor on the alert; there is no clear diagnosis of illness, and yet the patient cannot feel well. Here especially the doctor must eliminate tuberculosis as a cause before he can feel happy. When the patient complains only of increasing tiredness, failing appetite, scanty or suppressed menstruation and so on, this is the stage where the general practitioner has to be on his guard.

Routine Investigation

Each doctor adopts his own particular routine. In a country practice people much prefer to stay as far as possible under the care of their own general practitioner, and I firmly believe that it is one's duty to investigate the case oneself. After all, it is quite within the province of the family doctor, and he should not have to refer every slight suspicion to the local chest clinic. In the first place, it is something which a general practitioner can do just as well as the expert, so that it relieves the specialist of routine trivialities. Secondly, it is much kinder to the patient, for the word " tuberculosis " need never be mentioned, whereas the clinic used to be only too clearly labelled. Even now, the association lingers. My early zeal for truth-at-any-price made me condemn vague phrases, like " a little bronchial catarrh," which I heard older and wiser men using to patients when suggesting an X-ray or a sputum examination. Now I find myself using them when speaking to the apprehensive patient. *For in his search for a real disease he must not infect the patient with an imaginary one.* Fortunately a routine X-ray nowadays is becoming a commonplace to the general public; but there was a time when it was a certain match to light up any latent anxiety neurosis, and the patient would regard an appointment with the radiologist as the kind but certain way of sealing his fate.

A reasonable routine for investigation by the general practitioner is as follows: An X-ray of the chest and a sputum examination. (These facilities should undoubtedly be open to all general practitioners all over the country. The tendency is to increase them universally, and it will obviously greatly relieve the burden on the out-patient departments and the clinics.) If the patient is sufficiently intelligent, he should keep a temperature chart for two to three weeks. On this same chart the weekly weight can be recorded preferably by the doctor himself in his own consulting room, and of course he must do a routine physical examination of the patient.

I have never used E.S.R. estimations as a routine, so that I am not competent to say how far judgment can be placed upon them, neither do I do any blood examination (except a hæmoglobin estimation when there is pallor). But I feel strongly that the general practitioner should include both in his routine, either by undertaking them himself or by referring them to the laboratory.

If all these routine procedures return normal results, then the general practitioner can keep his mind easy, provided he sees the patient again at monthly intervals until he or she appears to have regained his normal state of well-being; if he does not, then one can repeat any appropriate test.

But where there is any doubtful or positive finding, then one should refer the patient to the expert. I estimate that this preliminary sifting only brings up about one doubtful case in twenty-five to thirty, and even of these few turn out to be true cases of pulmonary tuberculosis, the remainder are found to be suffering from some other pulmonary abnormality—a mild catarrhal bronchitis, an incompletely resolved virus infection, etc.

Domiciliary Treatment pending Admission

Ideally, as soon as early diagnosis is made, the patient ought to be admitted for sanatorium treatment. The shortage of nurses and of beds, however, makes this impossible at present, and the general practitioner must be prepared to carry out any intermediate treatment suggested by the chest physician. The powerful action on the tubercle bacillus of streptomycin and PAS puts a weapon into the hands of the doctor which can be used very successfully to check the spread of the infection while the patient is waiting for admission. The general practitioner cannot often give an injection once or twice a day for any length of time, but this may safely be delegated to most district nurses. It is the doctor's responsibility to see that she really understands the procedure and the technique, but the team of three can, and does, carry out very successful treatment if they work well together.

Contacts

It is the general practioner's duty to see that every precaution is taken to prevent the spread of the disease to contacts in the house. This is also a statutory duty of the health visitor, and there should be much closer co-operation between the health visitor and the family doctor. I am afraid that they normally see little of each other, and one comes across cases where they regard each others work as an interference with their own. There must be far more emphasis on their mutual dependence, particularly during the impressionable years of training on both sides. To my mind, it is in this question of " contacts " that the service could be improved, for there is not yet clear and happy collaboration between the three people concerned—the general practitioner, the health visitor and the chest physician; there can either be neglect or friction as a result, usually because the three do not know each other personally.

Siblings

In every practice there are families which are prone to tuberculous infection. I remember three such families where every young adult contracted the disease in turn, and there are now only two of the original twelve alive. The family doctor is the only man who can keep trace of the siblings, and quietly keep an eye on them as they in turn marry and start families of their own.

Under industrial conditions, the works doctors should be included, and the general practitioner should be given the results of any abnormal conditions found by mass radiography.

Bovine Tuberculosis

There remains the question of bovine tuberculous infection. Here the local doctor must make it his business to know something of the conditions under which the local milk is produced. During the war, tuberculous glands developed in an alarming way in the small evacuees after a few months spent in the country. A possible explanation was that many of them had up till then drunk nothing but pasteurised milk; they had not been gradually submitted to the natural vaccination by the occasional ingestion of the tubercle bacillus, and their first dose was too much. At the same time, there were two or three cases of tuberculous meningitis from the same cause. The general practitioner has to decide what to advise the young mothers. Advice which is usually given and which seems reasonable is to suggest that if the milk supply is not from a T.T. herd, or pasteurised, or both, then it should be scalded, until the child is at least three years old. Somewhere between the age of three and five the mother can be told to relax the precaution gradually, otherwise the child will receive no active immunity at all, but it is a very difficult problem, and each doctor has to solve it according to his own experience and knowledge of local conditions. I feel that general practitioners as a whole often lack expert direction in the advice they give to mothers, and this can only be remedied by a much closer personal contact between them and the local chest physician. Very often they hardly meet or even know each other by sight, and it is to be hoped that in the future each will realise his dependence upon the other and will make it his duty to work much more closely together.

The general practitioner has also to be on the look-out for early bone infection. There are so many minor sprains in children and young adults that he cannot X-ray every single one; but here again one must always say to oneself, " Could this be an early tuberculous infection ?" and if after a few days' rest it has not completely recovered, open access to the local radiologist is essential. A routine temperature chart and a Mantoux test will help to decide whether he should refer the case to the chest physician for his opinion.

I have tried to outline the place of the general practitioner in the tuberculosis service. Without his intelligence and alertness, the chest physician will receive nothing but cases so far gone that they are either hopeless or permanently maimed. Early and accurate diagnosis remains the most important part of the family doctor's work, and he must be helped with every facility within his proper range. He is the first line of defence against ill-health in any form, and as tuberculosis ranks so high amongst the diseases of his patients it must never be absent from his thoughts.

THE INCIDENCE OF TUBERCULOSIS IN SCHOOLCHILDREN

By Basil Wolman

From the Department of Child Health, University of Manchester

Since the beginning of the present century, when it was uncommon to find a child reaching adult life without being infected with tuberculosis, there has been a steady decline in the incidence of this disease in the childhood population of this country.

The morbidity rate of tuberculosis in adults, the principal source of infection in children, has fallen, due to improved measures of prevention, greater care of patients, increased education of the public and a change for the better in the general social and economic conditions of the country. However, the number of cases of miliary tuberculosis and meningitis occurring in children of pre-school age is still extremely high, and although a routine medical examination of children is carried out at the various school clinics, no attempt is made to determine the incidence of tuberculous infection in these children. Furthermore, now that the vaccination, with B.C.G., of large numbers of tuberculin-negative reactors is possible, it is important to know the magnitude of the problem and the percentage of the childhood population that might be involved.

In the present survey 1,076 local healthy children attending three "primary" schools in the vicinity of the university were given a complete clinical examination. The area is a typical working-class district within two miles of the city centre. The incidence of tuberculous infection in these children was determined by means of a tuberculin skin test and by mass miniature radiography of their chests. Undoubtedly the most accurate estimate is obtained from a tuberculin test survey, and the Mantoux test is the most reliable.

The Mantoux test was carried out in all children by the intradermal injection of 0·1 c.c. of $\frac{1}{1000}$ old tuberculin—*i.e.*, 0·1 mgm.—into the flexor surface of the arm previously cleaned by methylated ether. The reaction was read after forty-eight hours and was recorded as positive when there was a raised, red, circular or oval area more than 1 cm. in diameter, the centre being more raised than the periphery. Most of the children were tested in the schools with the co-operation of the head teacher, but some tests and readings had to be done in the homes, owing to a high absentee rate for trivial reasons.

All the children had their chests X-rayed by the mass miniature radiography procedure, any child with a suspicious shadow being recalled for larger films.

Of the 1,076 children tested, whose ages ranged from 4 years to 15 years, 200—*i.e.*, 18·5 per cent.—gave a positive reaction. The number of positive reactions among the boys (97 out of 540) was approximately the same as that for the girls (103 out of 536). As would be expected, there was a gradual increase in the number of positive cases with increasing age. Only 4 per cent.

of 91 four-year-old children gave a positive reaction, while, by the age of 12 years, 32 per cent. showed a positive Mantoux test (Table I).

TABLE I.—INCIDENCE OF POSITIVE MANTOUX TESTS WITH VARYING AGE GROUPS

Age	*Boys*		*Girls*		*Total*		
	No. Tested	*No. Positive*	*No. Tested*	*No. Positive*	*No. Tested*	*No. Positive*	*%age*
3	24	0	22	0	46	0	0
4	48	1	43	3	91	4	4·3
5	65	11	48	5	113	16	14·1
6	63	5	60	8	123	13	10·6
7	48	8	50	8	98	16	16·3
8	65	11	58	10	123	21	17·0
9	43	8	51	10	94	18	19·1
10	36	5	59	12	95	19	17·8
11	32	11	45	12	79	23	29·8
12	38	10	32	13	70	23	32·8
13	44	16	30	8	74	24	32·4
14	30	9	34	11	64	20	31·2
15	4	2	4	3	8	5	62·5
Total	540	97	536	103	1,076	200	18·5

The conclusion to be drawn from these figures is that at least 18·5 per cent. of local healthy school children have, or have had, a tuberculous infection. The term " at least " is used deliberately, for there may be no reaction to the Mantoux test, even in the presence of the disease, in the period before allergy develops—*i.e.*, usally four weeks after the primary infection—and furthermore, in the first few weeks after allergy has developed the reaction may be specific, human infection not responding to bovine tuberculin or *vice versa;* but apart from these considerations, both rarely encountered, and considering that no child was febrile at the time of testing, a negative test definitely excluded tuberculosis.

Another obvious consideration is the large number of children who have been infected and yet have never really been ill nor have they shown symptoms suggesting any such infection. Thus, primary tuberculosis in *childhood* can well be regarded as a mild disease with a normal tendency to recovery, but it is not known how many children of comparable age groups have actually died of tuberculosis due to the spread of the disease from a primary complex to a rapid miliary or meningitic end, although it is in infancy that there is the greatest danger from these evils.

The declining incidence of tuberculous infection to which reference was previously made can be seen (Table II) when the above figures are compared with those of Mantoux, who, in 1909, found that 50 per cent. of the children he tested in Paris were positive by 5 years of age and 80 per cent. by 12 years. Similarly, about the same period, Hamburger and Monti in Vienna found almost 100 per cent. positive reactions in children by the age of 14 years.

In more recent times in this country, Gaisford in 1930 found 21·5 per cent. positive out of 400 children under 14 in East London. Dow and Lloyd

in 1931 found out of 777 children, 23 per cent. positive from 0 to 4 years and 33 per cent. positive from 5 to 9 years. Hart in 1938 found 19 per cent. positive reactions among 536 children, and Bradshaw in 1939 reported 24·8 per cent. positive among 3,010 children. From Cardiff, Watkins reported in 1945 a positive incidence of 22 per cent. among 1,249 children. A similar drop in the incidence of the disease in American school children was reported by Myers, who found 47 per cent. positive in 1926, 19 per cent. in 1936 and only 7·7 per cent. in 1944.

TABLE II.—COMPARISON OF VARIOUS MANTOUX SURVEYS

Name	*Place*	*Year*	*No. Tested*	*% Age Positive*
Mantoux	Paris	1909	—	80
Hamburger and Monti	Vienna	1909	—	100
Gaisford	London	1930	400	21·5
Dow and Lloyd	Brompton	1931	777	23
Hart	London	1938	536	19
Bradshaw	London	1934-9	3,010	24·8
Watkins	Cardiff	1945	1,249	22
Myers	Minneapolis, U.S.A.	1926	—	47
Myers	Minneapolis, U.S.A.	1936	—	19
Myers	Minneapolis, U.S.A.	1944	—	7·7
Heimann and Simon	Bournemouth	1945-9	3,269	18·2
Present survey	Manchester	1949-50	1,076	18·5

Although the present study deals with a working-class population in an industrial area, the figure of 18·5 per cent. tuberculin positive compares very favourably with the 18·2 per cent. positive out of 3,269 children reported by Heimann and Simon from Bournemouth, a good-class residential area, in 1950.

The Mantoux test gives no help in deciding whether infection is active or healed, recent or old, extensive or localised, and although attempts have been made to correlate the size of the reaction with the extent of the lesion, the relationship has no practical significance. However, as a result of the mass miniature radiography and follow-up X-ray survey, radiological abnormalities were found in 27 cases only, and of these 4 cases showed abnormal cardiac outlines and 1 case showed a congenital defect of the ribs. Thus, 22 cases only showed abnormalities in the lungs on radiographic examination. Of these, 5 were reported as showing a healed primary focus. All were Mantoux positive, and on testing each had a normal sedimentation rate. Eight others were reported as having calcified nodules in the lung field or at the hilum, but only 5 out of 7 of these gave positive Mantoux tests using $\frac{1}{1000}$ tuberculin. Nine other cases showed areas of opacity at the base of a lobe, and all but 2 of these were Mantoux positive, but, judging by the radiological appearances, clinical examination and normal sedimentation rates in these 7 children, it was considered unlikely that the condition was tuberculous in origin.

The few radiologically abnormal lungs—22 cases—is in striking contrast

to the 200 positive Mantoux reactions. Sweaney in 1941 estimated that not more than 20 per cent. of primary complexes were visible radiologically, the remainder being of insufficient density or hidden by heart or diaphragm. It is therefore important to stress the value of the Mantoux test as a diagnostic weapon.

Apart from the first few weeks after allergy has developed, the tuberculin reaction is non-specific in regard to the type of tuberculin used. In other words, children infected with either human or bovine tubercle bacilli will give a positive test to either tuberculin. It is therefore difficult, particularly in older children who have almost certainly drunk unboiled milk at some time or other and mixed with numerous people, to trace the source of infection. In younger children infection is nearly always due to the inhalation of tubercle bacilli by droplet spread from an adult with open disease, usually a parent or someone living in the home. An attempt was therefore made to try to establish the source of infection in the younger children—those 4 and 5 years of age—of the twenty children with a positive Mantoux test in this age group. There was a definite family contact—parent, grandparent or aunt—in seventeen instances. In only three cases could no definite family contact be established. This again stresses the danger to the child from an adult with open disease in the home. Careful inquiry in these cases could not elicit any definite history of illness that might be attributed to the primary infection—nor was there any evidence suggesting a possible erythema nodosum or phlyctenular conjunctivitis.

All the 876 children with a negative tuberculin test were re-tested three to six months later, using the same strength tuberculin (Table III). Of these 876, 42—*i.e.*, 4·7 per cent.—were now found to give a positive reaction. This conversion had occurred in 20 out of 443 boys and 22 out of 433 girls.

TABLE III.—NUMBER OF MANTOUX CONVERSIONS AFTER THREE MONTHS

Age	*Boys*		*Girls*		*Total*		
	No. Tested	*No. Positive*	*No. Tested*	*No. Positive*	*No. Tested*	*No. Positive*	*%age*
3	24	0	22	0	46	0	0
4	47	0	40	0	87	0	0
5	54	0	43	0	97	0	0
6	58	1	52	6	110	7	6·3
7	40	2	42	1	82	3	3·6
8	54	1	48	2	102	3	2·9
9	35	2	41	0	76	2	2·6
10	31	2	47	7	78	9	11·5
11	21	1	33	3	54	4	7·4
12	28	2	19	1	47	3	6·3
13	28	6	22	2	50	8	16·0
14	21	3	23	0	44	3	6·8
15	2	0	1	0	3	0	0
Total	443	20	433	22	876	42	4·7

Inquiry into the health of these 42 children during the period since the previous testing revealed no definite history of illness that might be attributed

to a primary infection. The erythrocyte sedimentation rate was ascertained in all these children. In only 8 out of 42 was an elevated value (over 10 mm. in 1 hour—Westergren) obtained, suggesting that although these children were symptom-free and showed no abnormality on clinical examination, some active process, presumably due to a primary infection, was taking place. In none of these 8 children was any abnormality detected on radiographic examination of the lungs.

However, 7 other children, whose Mantoux had become converted, showed radiological abnormalities in the lung fields. In each instance this appeared as a focus in the periphery of the lung associated with some enlargement of the hilar glands—a typical primary complex. The erythrocyte sedimentation rate was normal (10 mm. per hour or less) in all these children, and it was presumed that a primary infection had taken place during the previous three to six months without provoking any symptoms and was now in a non-active phase.

An attempt was made to trace the source of infection in those children aged 6 years or less, but in only three instances out of 7 children in this age group was a contact traced. In two cases the mother had been found to be suffering from the disease, and in the third case, a brother had returned home from a sanatorium in the period since the previous testing.

Summary and Conclusions

1. 1,076 local healthy school children were Mantoux tested and 200—*i.e.*, 18·5 per cent.—were found to give a positive reaction. Thirty-two per cent. showed a positive test by the age of 12 years.

2. The incidence of tuberculous infection is less than that reported earlier in the century, but is the same as that reported last year for a good-class residential seaside area.

3. Only 11 per cent. of the positive reactions showed any radiological abnormality in the lung fields.

4. The conversion rate within three to six months was found to be 42 out of 876—*i.e.*, 4·7 per cent.—but in only 8 was there any evidence of an active inflammatory process taking place at the time of testing, as shown by an elevated erythrocyte sedimentation rate. Radiological evidence of a primary complex, presumably non-active, was noted in 7 other children.

5. A primary tuberculous infection in *childhood* can be regarded as a mild disease with a normal tendency to recovery.

6. The diagnostic value of the Mantoux test is stressed. It is the most reliable method for recognising tuberculosis in childhood, and by its use in the routine testing of the younger age groups may help to spotlight the presence of a contact in the household.

This study was made possible through the courtesy of Professor F. C. Wilkinson of the Turner Dental School of the University of Manchester, who already had the children of the three schools concerned under observation for prevention of dental caries; by the co-operation of Dr. E. M. Jenkins, School Medical Officer for the City of Manchester; and by the help and co-operation of the head teachers and staff of the schools concerned. My thanks are also due to Dr. W. Lee and Dr. R. Walshaw of the Manchester Chest Centre for carrying out

the mass miniature radiography and to Professor W. F. Gaisford for helpful advice in the preparation of this report.

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SPONTANEOUS PNEUMOTHORAX RESULTING FROM PNEUMOPERITONEUM THERAPY

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Introduction

So far only four cases of pneumothorax caused by artificial pneumoperitoneum have been described in the British literature. Of these only three have been carefully documented. In North America a further nineteen cases are mentioned and seven have been described in detail.

The purpose of this communication is to report a fifth case in this country; to examine the relevant literature, and to discuss the etiological factors, since this complication appears to be occurring more frequently.

Pneumoperitoneum—its Complications

Pneumoperitoneum has been used for therapeutic and diagnostic purposes since 1895, and in 1909 Bainbridge reported on the use of intra-abdominal oxygen in four cases of tuberculous peritonitis. The use of pneumoperitoneum was, however, practically confined to radiology until 1934, when Banyai was able to publish a series of 100 cases of pulmonary tuberculosis treated by pneumoperitoneum, of which seven had a supplementary phrenic nerve interruption.

In 1921 Case questioned 223 American radiologists and found that of these only twenty-one had extensive experience of the procedure at that time. He listed the risks of the procedure as puncture of intestine, blood vessels or viscera, air embolus, peritonitis, superficial emphysema and cardiac failure.

Despite Banyai's communication, which included a precise account of the method of carrying out pneumoperitoneum treatment in tuberculosis, we find Johannides and Schlack proposing pneumoperitoneum and phrenic nerve interruption as " a new principle in the treatment of patients in whom artificial pneumothorax is unsuccessful because of extensive adhesions " as late as 1937. It is clear that the use of this procedure was not then widespread.

In 1939 Banyai and Jurgens described seven cases of mediastinal emphysema complicating pneumoperitoneum refills given by the subphrenic route, and in 1938 Stokes mentioned massive atelectasis, scrotal pneumocœle and atrophy of the diaphragm as new complications of treatment. He deduced the latter by observing that an artificial pneumothorax following on pneumoperitoneum resulted in unusual downward displacement of the diaphragm.

The first case of pneumothorax occurring spontaneously during the course of pneumoperitoneum therapy is that of Mellies in 1939. The patient was a young girl with advanced disease. Three weeks after the commencement of pneumoperitoneum a pneumothorax developed and the presence of a fistulous communication between pleura and peritoneum was clearly demonstrated, since fluid could be tipped from one cavity to the other.

PLATE I.

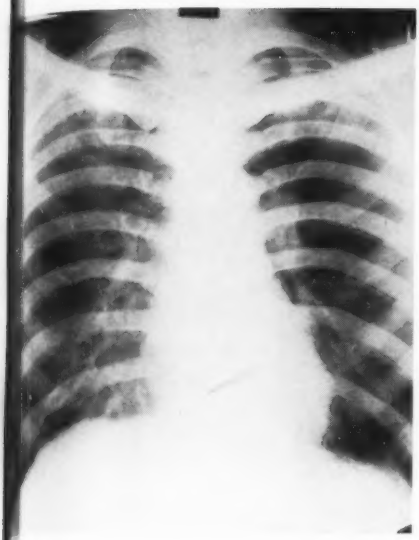

FIG. 1.—DISEASE BEFORE TREATMENT. SCATTERED INFILTRATION IN BOTH UPPER ZONES.

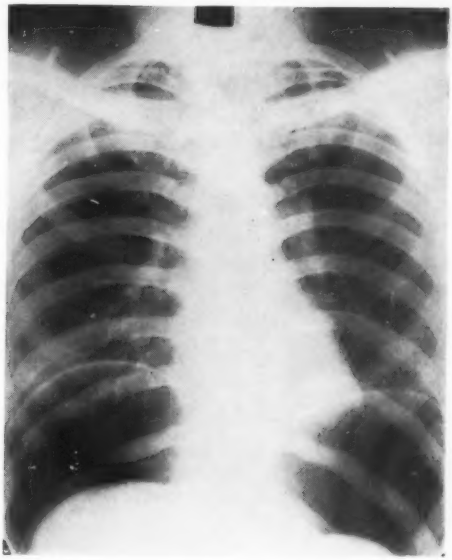

FIG. 2.—AFTER PNEUMOPERITONEUM INDUCTION.

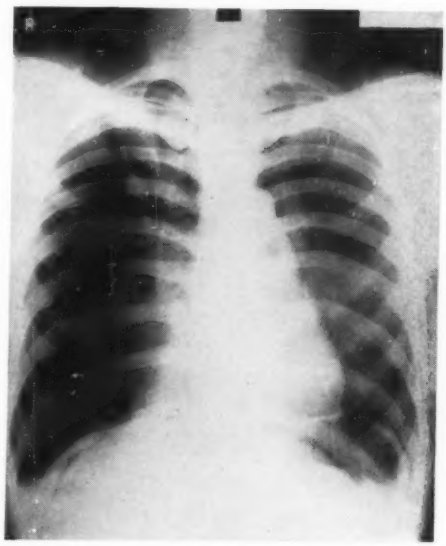

FIG. 3.—ON ADMISSION. FILM SHOWS A RIGHT PNEUMOTHORAX AND A SHALLOW PNEUMO-PERITONEUM.

To face page 30

In 1940 Banyai and Jurgens reported a second case, and in 1942 Newlyn Smith reported the first case in this country. It differed from all others in that the patient died of bilateral pneumothoraces following pneumoperitoneum induction during radiography.

In 1946 and 1949 Simmonds and Sita-Lumsden reported cases in this country, and in 1949 Yannitelli reported a fatal case from America.

Since then accounts have occurred in the literature with increasing frequency. In 1951 Wynn Williams reported a fourth case in this country, and Ross and Farber published a detailed account of three of their own cases, together with a brief account of twelve other cases they discovered on sending a questionnaire to sixty-one Californian chest physicians. (Unfortunately these are incompletely documented.) Repa and Jacobson (1951) have reported a further case.

A summary of the ten documented cases together with a further incomplete case of Cope's is presented in the Table.

The Present Case

A Pole, aged 28, a wood machinist, had been supervised as a contact since 1948. In September 1950, when he was symptom free, radiography showed scattered bilateral foci of infiltration in both upper zones with a small cavity in the left upper zone (Fig. 1). He discontinued work and was admitted to hospital in February 1951, when a pneumoperitoneum was induced (Fig. 2). After three months' treatment with bed rest and P.A.S. he was discharged in April, 1951. No phrenic nerve crush had been carried out. He made satisfactory progress resting at home and the cavity disappeared. Refills of 1,000 c.c. were given weekly and customary pressures were +9 cm. water before refill and +10 cm. after refill.

On 13.7.51 he had a further completely normal refill, but on returning home he assisted his wife in carrying a pram up fourteen steps.

During the afternoon of the following day he was seized by pain in the right chest and noticed slight dyspnœa when pulling on his trousers and straightening from the semi-erect position. The dyspnœa gradually increased during the following ten hours, and on the following day (15.7.51) he was admitted to hospital.

On examination he was dyspnœic at rest and the signs of a right pneumothorax were present. There was no clinical evidence of pneumoperitoneum. Radiography showed a deep pneumothorax on the right and a shallow pneumoperitoneum (Fig. 3). Neither clinically nor radiologically was there any evidence of mediastinal emphysema, and leading questions regarding this condition were unfruitful.

Aspiration of 800 c.c. of air from the pleural cavity caused disappearance of dyspnœa. Tomography of the diaphragm (1 cm. sections) showed no evidence of bulla formation on the pleural surface.

On 24.7.51 thoracoscopy was undertaken by Mr. A. H. M. Siddons to determine the divisibility of pleural adhesions and to find the site of air leakage from the peritoneum. Inspection of the right pleural cavity showed two adjacent blackened areas interrupting the pleural covering of the tendinous

SUMMARY OF AVAILABLE DATA FROM 12 CASES OF PNEUMOTHORAX COMPLICATING PNEUMOPERITONEUM

Author	*P.P. Established*	*Time after Refill*	*Phrenic Interruption*	*Phrenic Established*	*Demonstrated Pathology*	*Precipitating Factor*	*Side*	*Opinion of Authors*	*P.P. Pressures*	*Disease*	*Age*	*Sex*	*Pneumoperitoneum Continued?*
Mellies	3 weeks	1 day	Yes	5 months	—	—	R.	—	+4 but previously +10	Advanced	Young	F.	—
Banyai and Jurgens	12 months	1½ hours	No	—	—	Resting	R.	Mediastinal emphysema	+12	Advanced	53	M.	—
Simmonds	8 months	—	—	—	—	—	R.	Mediastinal emphysema	+10	—	29	F.	—
Yannitelli *et al.*	10 months	8 days	No	—	Diaphragmatic rupture. Diaphragmatic blebs seen on radiography	Laughter	R.	Diaphragmatic rupture	—	Mod. advanced	16	F.	Patient died
Ross and Farber	(1) 14 months	—	No	—	—	Defæcation	R.	Congenital defect	+12	Left upper lobe only	24	F.	Yes, with recurrence of pneumothorax
	(2) 4 months	—	Yes	4 months	—	Bending and lifting	R.	Congenital defect	+12	Mod. advanced	58	M.	Yes, successfully
	(3) 16 months	5 days	No	—	—	—	R.	Congenital defect	+7	Mod. advanced	20	F.	Yes, after pleurodesis
Cope	—	—	No	—	Diaphragmatic blebs seen at thoracoscopy	—	R.	—	—	—	—	M.	No
Repa and Jacobson	15 months	13 days	Yes, but regenerated	13 months	Diaphragmatic slit and blebs seen at thoracoscopy	Bending in bed	R. R.	Diaphragmatic rupture	+12	Mod. advanced	22	M.	No
Wynn Williams	6 months	Hours	Yes	4½ months	? Blebs on radiography. Possible diaphragmatic rupture seen at thoracoscopy	Coughing	R.	Diaphragmatic rupture	+12	Mod. advanced	44	M.	—
Sita-Lumsden	Same day	Immediate	Yes	8 days	Blebs seen at thoracoscopy	None	R.	Patent pleuroperitoneal canal	+5	Mod. advanced	38	F.	Yes, with recurrence of pneumothorax
Present Case	4 months	1 day	No	—	Diaphragmatic lesions seen	Sudden movement	R.	Diaphragmatic rupture	+10	Early	29	M.	Yes, successfully

— — No Information

part of the diaphragm. These areas were ¼ cm. and ½ cm. in length and roughly linear. They were situated 1 inch lateral to the vertebral column and 1 inch behind its anterior border. Some very small bubbles were present in the trace of pleural fluid in this region, but their site of formation could not be observed. It was considered that the darkened patches were definitely abnormal and suggested a small traumatic lesion, almost certainly the site of the air leak from the peritoneum. In addition there was a small patch of gelatinous grey-yellow exudate just posterior to the lesion. Division of adhesions was successfully carried out without complication.

On 2.8.51 and 7.8.51 1,000 c.c. refills were given into the peritoneal cavity as an experimental measure and careful screening has revealed no evidence of leakage. The pneumoperitoneum has now been abandoned.

Discussion

The pathology of this condition has been principally a matter for speculation until recently. When Mellies reported the first case of right-sided pneumothorax complicating pneumoperitoneum he did not discuss its etiology, but Anderson and Winn (1945) imply that tuberculous ulceration of the diaphragm was responsible. Banyai and Jurgens (1940), Simmonds (1946) and Loughead (1950) believed that the air tracked into the mediastinum via the aortic or œsophageal hiatuses, causing mediastinal emphysema and subsequent rupture of air into the pleural cavity. This theory does not explain how the peritoneum about the hiatuses is crossed nor why all the incidents have been right-sided. It also overlooks the fact that the pain and signs of mediastinal emphysema have been absent from all the reported cases. It should be noted that in mediastinal emphysema complicated by pneumothorax in non-tuberculous subjects the incidence of pneumothorax is equal on both sides (Dickie, 1948). And in addition, if we exclude Simmonds' controversial case, no case of pneumothorax has been described in mediastinal emphysema complicating pneumoperitoneum (Banyai and Jurgens, 1939; Meyer, 1949; Keers, 1948).

Examination of the literature gives one the impression that two types of case exist—those which develop immediately after the induction of pneumoperitoneum and those in which the pneumothorax occurs some months after induction. The former group consists of the cases of Newlyn Smith and Sita-Lumsden, which are probably due to a congenital abnormality, and all other cases fall into the second group ,which appears to be traumatic in origin. Mellies' case belongs to the latter group, but is exceptional in that the pneumothorax occurred only three weeks after induction.

The case described by Newlyn Smith was fatal and is the only example of a left-sided pneumothorax. (Right-sided pneumothorax was also present.) At autopsy a number of small holes were present in the diaphragm connecting the peritoneum and the pleural cavities. The patient did not have tuberculosis and it is fair to assume that the holes were congenital in origin. Sita-Lumsden has produced convincing evidence in favour of the presence of a patent pleuro-peritoneal canal in his case, although in common with the traumatic cases the pneumothorax was right-sided.

In 1947 Mitchell mentioned diaphragmatic rupture as a complication of

pneumoperitoneum, but did not express any view as to the etiology, although he believed that diaphragmatic atrophy occurred in pneumoperitoneum. The findings in the present case and the evidence presented by other authors make it extremely likely that traumatic rupture of the diaphragm is responsible for the bulk of cases of pneumothorax complicating pneumoperitoneum.

An autopsy in the case described by Yannitelli showed a diaphragmatic opening connecting the peritoneum and pleura. This opening was situated between two muscle bundles, and the description of the histology makes it clear that the lesion was traumatic in nature and that aseptic healing was in progress in the vicinity of the opening. Repa and Jacobson saw a postero-lateral slit in the diaphragm at thoracoscopy, and in Wynn Williams' case a bright red area was seen in the diaphragm, antero-lateral to the vertebral column. A lesion suggesting organising blood clot was seen in the present case.

In addition a cyst consisting of normal diaphragmatic tissue was seen on the pleural surface of the diaphragm in Repa and Jacobson's case, and similar blebs were seen on thoracoscopy by Cope, who observed that they varied in size with respiration. Yannitelli and Repa and Jacobson also saw these blebs in the radiograms of their cases, and Wynn Williams states that he saw two rounded shadows in relation to the right diaphragm in his case.

Laird (1945) was the first person to record the occurrence of these blebs at thoracoscopy during pneumoperitoneum treatment, and he expressed the view that their rupture might give rise to pneumothorax. On the evidence before us, such would indeed seem to be the case.

It is presumed that as a result of continuous stretching of the diaphragm by the pneumoperitoneum a hernial sac consisting of peritoneum, pleura and thinned out diaphragmatic tissue bulges into the pleural cavity, and that it is the rupture of this under added stress which gives rise to the pneumothorax. In this connection it should be noted that high pressures were carefully avoided when pneumoperitoneum therapy was first introduced (Banyai, 1934). Stokes actually specified that the pressures must not exceed +6.

At the present day it is often the aim of treatment to exceed such pressures, and the use of abdominal binders may increase the subdiaphragmatic pressure even further.

Macklin (1937) in the course of experimental work on animals pointed out that pleural blebs are unidentifiable after rupture. This would explain why no bulla is apparent at the actual site of rupture but may be seen in neighbouring areas of diaphragm.

In the ten cases in which we have information only four had undergone supplementary phrenic nerve interruption, and in one of these regeneration had taken place. It is therefore evident that this manœuvre plays no part in predisposing towards diaphragmatic rupture.

No really satisfactory explanation has yet been put forward to elucidate the exclusive right-sided occurrence of the spontaneous pneumothorax. Ross and Farber suggest that the removal of the support of the liver may unmask a diaphragmatic deficiency, but this cannot explain why no case of unilateral left-sided pneumothorax ha occurred.

It is somewhat easier to understand why this syndrome is occurring with increasing frequency. Pneumoperitoneum is now much more widely used than

ten years ago, and if this trend increases more instances of pneumothorax may be anticipated.

At first pneumoperitoneum was used only for advanced cases of pulmonary tuberculosis when nothing else could be done (Moyer, 1949; Banyai, 1938; Vajda, 1933), but now the scope of the procedure has widened to embrace predominantly unilateral exudative disease as a preliminary to further collapse treatment. It is not infrequently used where pneumothorax has not been attempted, and under these circumstances the pleural space may be intact. It is in such cases that pneumothorax can occur, and the absence of a pleural space in the advanced cases originally treated may provide a second explanation for the former rarity of this complication.

A third factor arises from the use of pneumoperitoneum in early cases of tuberculosis, for when these cases become ambulant they are likely to perform acts which will involve added pressure on the under surface of the diaphragm. It will be noted that in the seven cases in which this information is available six were making movements involving an increase in the intra-abdominal pressure (coughing, bending, laughing).

Measurements undertaken on the present patient have shown that during the act of coughing the intra-abdominal pressure rises by as much as 15 cm. of water above the intra-pleural pressure, whilst during prolonged straining the pressures differ by only 4 cm. of water. It is therefore evident that sudden violent motions are the most likely to cause diaphragmatic rupture.

Conclusions

Despite its incidence in persons already suffering from severe respiratory disease, pneumothorax complicating pneumoperitoneum does not have a high mortality. Out of twenty-four cases which are now recorded only two have been fatal (Smith, 1943; Yannitelli, 1949). Ross and Farber have shown that once this complication has occurred further attempts to continue the treatment are likely to be fruitless. In the present case no recurrence of leakage occurred during one week in which pneumoperitoneum was recommenced.

If air containing bullæ are seen on the pleural surface of the diaphragm during pneumoperitoneum therapy, they should be regarded, in the absence of mediastinal emphysema, as possible precursors of diaphragmatic rupture, and the supervision of the case should be modified accordingly. It must, however, be recognised that such blebs have occasionally been observed without rupture, and here the pathology probably differs from that described above, although its nature remains a matter for speculation (Sita-Lumsden, 1949; Banyai and Jurgens, 1939; Mitchell, 1947).

Summary

Leakage of air from a pneumoperitoneum to the pleural cavity is a complication which is increasing in frequency, but which has a low mortality rate. It is suggested that this occurrence is attributable to the increase in use of pneumoperitoneum, particularly in cases where the pleural cavity is not obliterated and the patient is ambulant.

Phrenic nerve interruption does not appear to predispose to this accident.

Diaphragmatic bulla formation has been observed in some of the reported cases, and probably this represents a stage in the pathology of diaphragmatic rupture.

On available evidence the latter must be considered the commonest cause of spontaneous pneumothorax in pneumoperitoneum, although a small proportion of cases may be due to a patent embryological communication between the peritoneum and the pleura.

We wish to thank Dr. Wallace Graig for the information about the patient while initially under his care, and also Mr. A. H. M. Siddons for his report on the thoracoscopy. The patient was admitted as an emergency under the care of Dr. A. L. Punch, to whom we are indebted for permission to publish the case report.

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PULMONARY ADENOMATOSIS

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The purpose of this paper is to report three cases of pulmonary adenomatosis, all diagnosed at autopsy. They were found during a period of five months by a hospital group serving a population of approximately 150,000. Either this is an unusual coincidence or, as Davis and Simon (1950), who saw five cases in one year at St. Louis, Missouri, have suggested, this disease is more frequent than is generally thought. Up to the present time only isolated cases have been reported in England. A number of detailed articles on pulmonary adenomatosis have appeared within the last ten years, and we are therefore outlining only brief case histories and pathological reports on these three patients.

There is no unanimity of opinion regarding the histogenesis of pulmonary adenomatosis. Swan (1949), who reported nine cases occurring among 900 neoplasms of the lung, accepted the following criteria of diagnosis: (1) alveolar cellular proliferation characterised by the appearance of tall columnar mucus-producing cells; (2) absence of an intrinsic tumour of the bronchial tree; and (3) absence of a primary adenocarcinoma of any other part of the body. The demonstration of mucus has not been regarded as obligatory by many authorities, particularly as this substance varies in composition and cannot be demonstrated by a single stain. Davis and Simon (1950), for example, only noted mucinous secretion in three of their five patients. Mucus was seen in each of the cases described here.

Case Histories

Case 1

A female aged 41 was first seen on October 18, 1949. She was an Estonian, which caused difficulty in obtaining an accurate history, but she stated that she had had a left-sided pleurisy about fifteen years previously, and had suffered from left-sided pneumonia on at least four occasions. A radiograph had been taken during 1948; since which time she had suffered from increased cough, sputum and shortness of breath on exertion. There had been no hæmoptysis or loss of weight, but for about ten days there had been pyrexia up to 100° F. in the evening. Physical examination showed a woman of average habitus, looking less than her age. There was no cyanosis, dyspnœa at rest or clubbing of the fingers. The heart and trachea were much displaced to the left, and the left side of the chest was flattened and moved little on respiration. There were no abnormal physical signs over the right side of the chest except a few rhonchi, but the left side was dull to percussion and bronchial breath sounds and many râles were present both anteriorly and posteriorly. A radiograph (Fig. 1) confirmed the cardiac displacement and showed fairly extensive shadowing over the right lung, most marked in the middle zone, but also extending into the lower and slightly

into the upper zones. The whole left lung field was opaque, but in the upper and middle zones there were rounded areas of increased translucency of the type seen in cystic bronchiectasis. A tentative diagnosis seemed to lie between pulmonary tuberculosis and bronchiectasis. Further investigations gave an Erythrocyte Sedimentation Rate (E.S.R.) of 40 mm. at 1 hour (Westergren), and numerous sputa were negative on direct smear and later on culture. A bronchoscopy was refused. A radiograph which had been taken on September 22, 1948, later became available. From this it could be seen that the shadows on the right side had extended considerably by October 1949, and that the collapse of the left lung had also increased. During the next few months successive radiographs showed little change, but in February and March 1950 there was moderate pyrexia lasting for about one month, and an X-ray film on March 28, 1950, showed an extension of the abnormal shadows on the right side. The weight had also fallen by 13 lb. Cough was very troublesome with 1 to 2 oz. of frothy sputum, which in May was stained with blood on several occasions. In June a further bout of pyrexia took place, and clubbing of the fingers was noted for the first time. The cough continued very troublesome, no more weight was lost, but dyspnœa was becoming her chief complaint. She was admitted to hospital in July, and was found to be running a moderate pyrexia. Penicillin was given and by the fifth day the temperature was normal. Her E.S.R. was 30 mm. at 1 hour (Westergren), numerous sputa proved negative for tubercle bacilli, but a moderate degree of hypochromic anæmia was present. She was discharged in August symptomatically improved, but admitted again in September 1950 with increasing dyspnœa, slight cyanosis, cough and frothy sputum. She was given streptomycin and paramisal sodium with some symptomatic improvement, but no change in the general condition or radiological picture. Discharged in October, she continued downhill and died on November 21, 1950.

Pathological Findings (*autopsy* 36 *hours after death*)

Macroscopic. At autopsy the left pleural cavity was completely obliterated by dense fibrous adhesions. There was a small quantity of clear yellow fluid in the right pleural space. The right lung was rather pale in colour, particularly in the two lower lobes, and felt curiously solid and indiarubbery. The posterior surface in the middle zone showed a grossly scarred and nodular arrangement. The scarring was relieved by areas of lung tissue which projected above the surface as smooth round nodules varying in size from 0·5 to 2 cm. across, the appearance of this area resembling that of post-necrotic cirrhosis of the liver with nodular hyperplasia. On section practically the whole of the lower and middle lobes and the lower part of the upper lobe were diffusely infiltrated by pale yellowish-white tissue which had a mucoid appearance and in the upper lobe was sharply demarcated from the remainder of the lung tissue. A careful dissection of the bronchi failed to show any localised tumour which might have been regarded as a primary. The left lung was smaller than the right and felt cystic on removal. On section large numbers of cystic spaces were found with dilated bronchi scattered throughout both lobes of the lung. The spaces had a smooth lining and in the larger ones, which were 3 or 4 cm. across, there was also trabeculation of the wall. There was no evidence of tumour in this lung. The hilar lymph nodes—in particular those at the bifurcation of the trachea—were greatly enlarged, and so to a lesser degree were those on either side of the trachea up to the level of the thyroid gland. On section they showed a greyish-white surface except where there were deposits of carbon. It was thought that

FIG. 1.—POSTERO-ANTERIOR RADIOGRAPH (18.10.49) SHOWING EXTENSIVE BILATERAL SHADOWING WHICH PROVED TO BE ADENOMATOSIS ON THE RIGHT SIDE AND BRONCHIECTASIS ON THE LEFT.

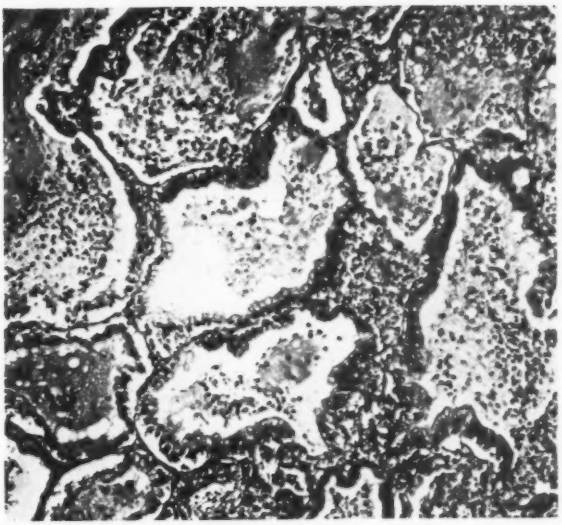

FIG. 2.—THIS SHOWS THE ALVEOLI LINED BY COLUMNAR EPITHELIAL CELLS AND CONTAINING A MUCOID MATERIAL AND NUMEROUS POLYMORPHONUCLEAR LEUCOCYTES. H & E × 96.

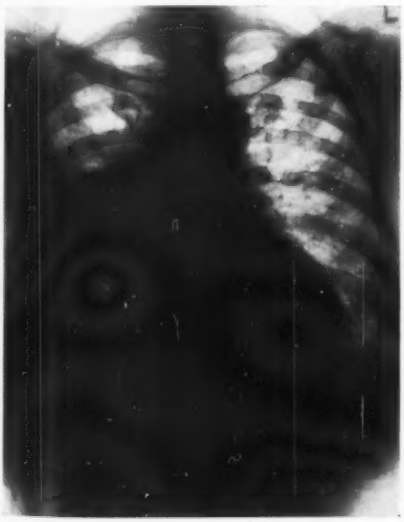

FIG. 3.—POSTERO - ANTERIOR RADIOGRAPH (4.9.50) SHOWING A RIGHT PLEURAL EFFUSION AND SLIGHT MOTTLING IN THE RIGHT UPPER ZONE. ROUNDED SHADOWS ARE ALSO PRESENT IN THE LEFT UPPER AND MIDDLE ZONES.

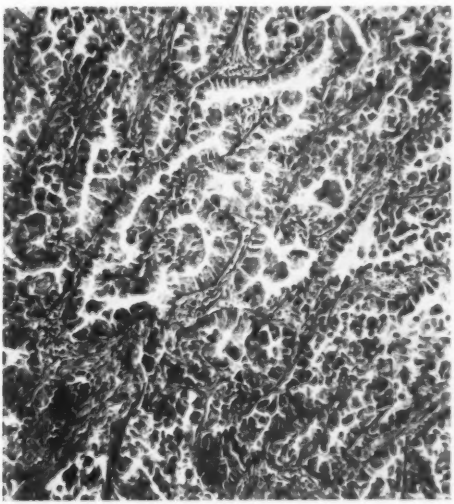

FIG. 4.—THE CLOSE INVESTMENT OF THE ALVEOLI BY COLUMNAR EPITHELIAL CELLS IS SHOWN HERE. PICRO-MALLORY × 96.

To face page 38

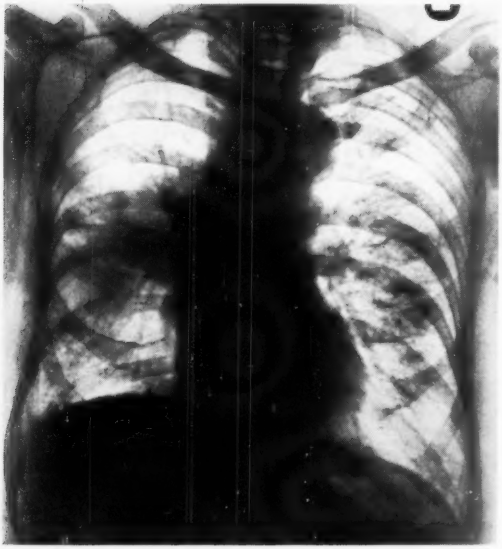

FIG. 5.—POSTERO-ANTERIOR RADIOGRAPH (8.3.51) SHOWING EXTENSIVE SHADOWING IN THE RIGHT MIDDLE ZONE AND A SMALL AREA OF SHADOWING IN THE LEFT MIDDLE ZONE.

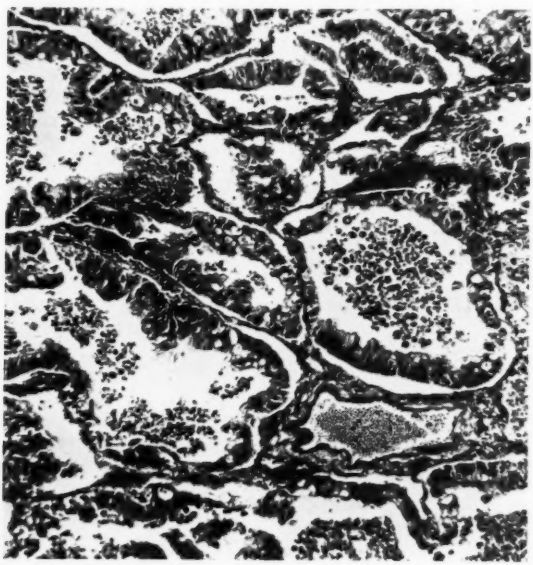

FIG. 6.—ALVEOLI ARE AGAIN SHOWN LINED BY COLUMNAR EPITHELIAL CELLS SOME HAVING VACUOLES WHICH STAIN POSITIVELY FOR MUCIN. THERE ARE POLYMORPHONUCLEAR LEUCOCYTES IN THE CENTRE OF THE ALVEOLI. MASSON'S TRICHROME STAIN × 96.

To face page 39

they were infiltrated by tumour, but histological examination showed that this was not so.

The other organs showed only evidence of chronic venous congestion. There were no deposits of secondary carcinoma in any of the organs. The bone marrow was, however, not examined.

Microscopic. In the right lower lobe the alveoli were partially or completely lined by a single layer of tall columnar epithelial cells (Fig. 2). These cells had large nuclei situated near the base of the cell—the cytoplasm being rather pale and often vacuolated. These vacuoles did not stain with mucin stains, but the free edge of the cells gave a strongly positive reaction with the periodic acid-Schiff technique and stained metachromatically with toluidin blue. The alveoli were filled with a pale pink staining homogeneous material faintly positive to mucin stains. The tumour cells often projected as small papillæ into the lumen of the alveoli, which in a number of instances contained considerable numbers of polymorphonuclear leucocytes. Reticulin and elastic tissue stains showed that the alveolar framework was preserved in the majority of the lung, but in some places there was considerable fibrous tissue reaction obliterating alveoli and tumour alike. There was little normal lung tissue remaining in the left lung, and the sections showed large tracts of vascular fibrous tissue with numerous cavities. These contained muscular tissue in their walls and were evidently dilated bronchi. Some of them were lined by remnants of bronchial epithelium, while others had granulation tissue in their walls. No tumour tissue was found in this lung. The lymph nodes showed reactive hyperplasia.

CASE 2

A female aged 73 was first seen on September 4, 1950, and admitted to hospital on September 29, 1950. She complained of dyspnœa increasing during the last nine months, with cough and a little sputum which had been blood-stained two weeks previously. For two months there had also been epigastric pain which had no relation to food, and some swelling of ankles in the evenings. Her history otherwise revealed nothing of importance, and there was no family history of tuberculosis. On examination she was pale, dyspnœic on slight exertion, with clubbing of the fingers. The heart was normal in position, the aortic second sound was accentuated, the blood pressure 175/85, and there was no œdema. Over the lower and middle zones of the right side of the chest anteriorly and posteriorly tactile fremitus was diminished, the percussion note dull, and the breath sounds very weak—signs typical of a pleural effusion. A radiograph taken on September 4, 1950 (Fig. 3), showed dense shadowing over the lower and middle zones of the right lung, and also slight mottling in the upper zone: over the left lung there were some rounded densities in the upper and middle zones. A penetrating film disclosed a dense opacity within the right lung, and a lateral view suggested that this was in the area of the middle and lower lobes. The chest was aspirated and 2 pints of straw-coloured fluid removed, which on pathological examination proved to have 18 per cent. polymorphs and 82 per cent. lymphocytes; no tubercle bacilli or other organisms were found, and culture yielded no growth. Her E.S.R. was 44 mm. at 1 hour (Westergren); a blood count was normal except for slight hypochromic anæmia. A fractional test meal showed slight hyperchlorhydria and the stools were negative for occult blood. Finally, after a number of negative examinations, a direct smear of the sputum was returned positive for tubercle bacilli. On this evidence and in the absence of any obvious malignant focus a diagnosis of

tuberculosis was made. In view of the positive sputum and her poor condition it was considered justifiable to try the effect of streptomycin and paramisal sodium, and this was started on October 10, 1950. There was no improvement and an intermittent temperature of 99° F. rising occasionally above 100° F. was unaffected. The further progress of the patient was steadily downhill. Dyspnœa was marked and little relieved by pleural paracentesis, which was performed on several occasions. The pleural fluid had become blood-stained by December and was re-accumulating rapidly. Numerous sputa had by this time been examined and cultured; all proved negative with the one exception previously mentioned. The patient's general appearance suggested malignancy, and this was considered in the differential diagnosis. She died on February 6, 1951.

Pathological Findings (*autopsy 20 hours after death*)

Macroscopic. There was a considerable quantity of clear yellow fluid in the abdominal cavity and a large amount also in the left pleural cavity. The right pleural space was largely obliterated by dense fibrous adhesions between the two layers of greatly thickened pleura. Such space as remained contained fibrinous material. The right lung was reduced in size and very firm to the touch, the surface being covered by thickened pleura. On section practically the whole of the lower lobe and the inner portions of the upper and middle lobes were infiltrated by greyish-white, rather soft opaque material. In the lower lobe this material was particularly obvious at the periphery, there being more apparently normal lung tissue in the centre of the lobe. The appearance suggested a tuberculous broncho-pneumonia, which was in fact the provisional diagnosis made at autopsy. The left lung showed numerous nodules of whitish material scattered irregularly through both lobes. Usually they had a central greyish area apparently due to carbon. These nodules, which varied from a few millimetres to about 1 cm. in diameter, were surrounded by fibrous tissue radiating from them. Where the nodules lay beneath the pleura, this gave rise to a puckered scar. The remainder of the lung tissue appeared normal. No primary tumour could be found in the bronchial tree. The lymph nodes of the mediastinum showed anthracosis but no evidence of tumour. No deposits of tumour were found in any other organs. The bone marrow was not examined.

Microscopic. In the right lung fibrosis was a much more noticeable feature of the histological material than in Case 1, but there were many areas which showed the characteristic appearance of thin alveolar walls lined by tall columnar cells with basal nuclei (Fig. 4). Some of these cells had a vacuolated cytoplasm, but the majority were eosinophilic. In this instance the vacuoles gave positive reactions for mucin, as did some of the material within the alveolar lumen. The alveolar pattern was shown to be intact in most of the lung by reticulin and elastic tissue stains. At the periphery of the lung, where it is adherent to the chest wall, lymphatic invasion was seen, and tumour was also found inside a small artery. Nodules of tumour tissue were found in the left lung, producing a histological picture identical with that of the primary growth, and once again there was a well-marked fibrous tissue reaction. The way in which the tumour cells lined the alveolar walls was particularly well seen in these nodules. No secondary deposits were found in the local lymph nodes.

CASE 3

A male aged 80 was admitted to hospital on March 6, 1951. He stated that for several months he had had increasing shortness of breath on exertion, marked loss of weight and progressive weakness. For the last seven days he had

experienced difficulty in swallowing food, and for about one month cough and sputum. On physical examination the heart apex beat was found 4½ inches from the mid-line in the fifth left intercostal space, and a systolic murmur was heard over the aortic area. Over the upper zone of the right lung anteriorly there was flattening, diminished movement, slight impairment of percussion note and weak breath sounds; posteriorly over the whole of the right side of the chest there were dullness on percussion, weak breath sounds and slightly increased vocal resonance. Apart from a right inguinal hernia no other abnormality was found except generalised wasting. A radiograph (Fig. 5) on March 8, 1950, showed some scoliosis, and a slightly raised right hemi-diaphragm with an obscured costo-phrenic angle; the heart was slightly displaced to the right, the aorta definitely enlarged. On the right side the interlobar septum was slightly thickened, and a moderately dense opacity occupied the middle zone, though spreading slightly above and below it. In the left lung a small area of increased shadowing was present in the middle zone. A diagnosis of carcinoma of the bronchus was made. His temperature, which had been normal, rose to 101°F. on the fourth day after admission, and two days later a pleural rub became evident over the middle and lower zones of the right lung anteriorly, and crepitations were heard over the same zones posteriorly. Penicillin was administered, but death occurred in coma in March 14, 1951.

Pathological Findings (*autopsy 24 hours after death*)

Macroscopic. A very emaciated elderly man. There was no free fluid in the abdominal cavity nor in the left pleural space. The right pleural cavity was largely obliterated by dense fibrous adhesions. The right lung was extremely heavy and on section found to be heavily and diffusely infiltrated by greyish-white tumour tissue. The whole of the lower lobe was involved, showing only a little normal lung tissue towards the centre of the lobe. The middle lobe contained a few rounded nodules of tumour tissue with the largest 1·5 cm. in diameter, while others were only a few millimetres across. In the upper lobe there was a rather diffuse involvement near the hilum and just above the interlobar fissure, but there was no other involvement of this lobe by tumour. There was thickening of the pleura and evidence of healed tuberculosis in the apex of this lobe. In the left lung there were nodules of whitish tumour a few millimetres to a centimetre across. They did not show any central grey areas and the degree of scarring was much less than in Case 2. There were several lymph nodes in the mediastinum showing involvement by tumour tissue, and one at the level of the thyroid cartilage on the right side contained tumour tissue. No primary tumour was found in the bronchial tree and the other organs showed no secondary deposits, but the bone marrow was not examined. The aorta showed the characteristic appearances of syphilitic aortitis.

Microscopic. Sections of the right lung (Fig. 6) showed a histological picture similar to Case 2. The alveoli were large and the neoplastic lining cells very big, often having a pseudostratified arrangement. Many of the cells had vacuoles giving a positive reaction with the periodic acid-Schiff routine and stained metachromatically with toluidin blue. The alveolar framework remained intact as shown by reticulin and elastic tissue stains, but fibrous tissue formation was marked in places. Many of the alveoli were filled with a purulent exudate. An interesting finding was that in some places the alveoli were lined by a single layer of cubical epithelial cells which undoubtedly possessed cilia. Every gradation between these cells and the fully developed

tumour cells could be found, and they were in some instances found lying in adjacent alveoli separated from one another by the alveolar wall. We were, however, not able to demonstrate continuity of the ciliated cells with the more obviously neoplastic cells. Lymphatic invasion by tumour was found but no blood vessel invasion. The tumour nodules in the left lung showed precisely the same features as the primary growth, but no ciliated epithelium was found. The local lymph nodes contained tumour tissue which closely mimicked that of the primary growth, with the exception that the septa supporting the growth were somewhat thicker than in the lung, where the alveolar walls provided the framework. The sections of the aorta confirmed the diagnosis of syphilitic aortitis.

Discussion

The clinical and radiological appearances of these three patients are typical of those hitherto described in that they were unalike, suggestive only in their variability, and undiagnosed before autopsy. We do not propose to enter the theoretical discussion as to the origin of these tumours, except to draw attention to the finding of ciliated cells in the third case, which were clearly very closely related to the tumour cells both as to position and appearance. This suggests that this tumour, at least, arose from bronchiolar epithelium rather than from a still disputed alveolar lining cell. Swan also found ciliated epithelium in his Case 3, but regarded it merely as evidence of origin from pluripotent parent cells.

The malignancy of these tumours is variable, and this is shown by the cases presented here. Thus Case 1 had radiological evidence extending over two years and at death the tumour was confined to one lung, while in the other two cases symptoms were of a year or less and the lesion was more widespread at autopsy. There are other cases in the literature in which distant metastases have occurred and these may be regarded as an expression of even greater malignancy.

It is fairly generally accepted that in most organs there is a range of tumours spreading from the quite benign at one end of the scale to the frankly malignant at the other, and that no sharp dividing line exists. We believe that much the same sort of situation exists with these tumours, but here the most benign of them lies towards the malignant end of the scale. On this account most of these tumours are to be regarded as carcinomas.

Summary

Three cases of pulmonary adenomatosis coming to post-mortem within five months from a population of about 150,000 are described clinically and pathologically. It is suggested that this disease is not so rare as is often thought.

Our thanks are due to Dr. J. W. Clegg for his confirmatory opinion on the pathological material, and to Dr. J. H. L. Easton for access to the clinical notes on Case 3.

REFERENCES

DAVIS, M. W., and SIMON, T. R. (1950): *Amer. Rev. Tuberc.*, **62**, 594.
SWAN, L. L. (1949): *Arch. Path.*, **47**, 517.

PNEUMOCONIOSIS IN BOILER SCALERS

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The occurrence of pneumoconiosis in boiler scalers was first recorded by Cooke (1930) and Williams (1931), but it was not well recognised until some twelve years later. One of us (L.D.) published a paper on the subject in 1943 and another the following year in collaboration with Hermon. Todd and Rice also reported a case in 1944 showing a miliary type of pneumoconiosis. Harding, Tod and McLaughlin published papers in 1944 and 1947 dealing particularly with the pathological aspect in two cases, and showed that the lung lesions were due to silicosis and pneumoconiosis respectively.

Cook, Kemp and Wilson (1945) reported negative findings in nine boiler-cleaners at a railway depot. The impression was given that boiler scalers generally are not at risk in their occupation, but we have seen many scalers who have been working at their trade for a long time in whom chest X-rays were negative. However, only positive radiological findings of pneumoconiosis are decisive in determining whether this disease is an occupational one.

Since 1943 we have examined a further number of boiler scalers, some with negative radiological findings and some with positive evidence of abnormalities in the lungs, and this paper is a review of 25 cases with positive radiological findings of whom 4 had carcinoma, 4 had tuberculosis, and 17 had manifestations of pneumoconiosis of various types.

Conditions of Work

It is important to mention that all the men seen by us had been or were scaling the boilers of ships, where work conditions are far from good, being in confined spaces, the air full of dust. Access to the interior of the boiler is through a narrow manhole which is also the only exit for the dust arising from scaling with hammer and chisel.

The worse the environmental conditions the greater the quantities of dust inhaled, chemical analysis of which has shown to contain free silica in harmful proportions and of critical particle size. Apart form scaling inside the boiler the flues (pipes) which cross its interior require to be cleaned by long wire brushes; the dustiest work is shovelling the flue dust from the back end of the boiler.

Scalers are therefore exposed to two types of dust—flue dust and boiler scale. Our analysis of the dusts is contained in the following report (D. J. T. B.).

The fine fractions, having particle sizes of less than 5μ, were separated by means of a current of air from a sample of boiler scale, flue dust and back

end flue dust, and their total and free silica contents determined. Total silica includes silicates.

	Total Silica Percentage	*Free Silica Percentage*
Boiler scale	14·5	1·1
Flue dust (sooty)	13·5	6·2
Back end flue dust	23·0	10·5

The figures for free silica were obtained by the method of Gurvits and Podgayts, after allowance had been made for the presence of silica in silicates which remained insoluble after the treatment. Its accuracy when dealing with these samples is not known, and probably the most reliable method is that of X-ray diffraction. The ratios of total silica : free silica are the same in the above two dusts. It may be noted that the total silica content of the ash of normal British coals varies from 25 to 50 per cent.

The proportion of silica in boiler scale is dependent upon the silica content of the feed water after any treatment for softening. The use of siliceous zeolites for this purpose, especially the synthetic type, may increase the silicate content of the raw water.

The above results show that the fine fraction of back end flue dust contains more silica than that of the boiler scale, but as workmen are always exposed to both, this distinction has no practical significance. The dusts also contain iron which may be inhaled.

Individual susceptibility to the development of pneumoconiosis in boiler scalers is noteworthy and is analogous to the apparent susceptibility among coal miners. Men working under seemingly identical conditions do not react uniformly, some remaining completely unaffected by the inhalation of the relevant dust.

Clinical Findings

The presenting symptoms in workers who are ill are not typical of occupational lung disease, and resemble the symptoms of other chest complaints encountered in patients referred to chest clinics. Productive cough and occasionally bloodstreaked sputum are common complaints, but no less frequent than among the adult population of an industrial city with climatic conditions such as exist in Hull. Dyspnœa is, however, the chief complaint, and may present itself in boiler scalers long before any radiological signs of pneumoconiosis appear. On the other hand, dyspnœa may be absent or not admitted even on direct questioning in boiler scalers who exhibit marked radiological signs of pneumoconiosis. There is a third group of patients with X-ray evidence of pneumoconiosis who may not complain of dyspnœa until many years later (the onset being sudden or more gradual), in spite of no obvious X-ray deterioration or progression of the disease. One has to be cautious in attributing dyspnœa to any single factor, especially in patients of advancing years.

The underlying cause of the dyspnœa has not yet been fully established. In many cases it is probably due to emphysema, which has been found in the few pathological reports available. Chest signs often conform with these findings. We have found almost invariably on clinical examination that there is impaired chest expansion and diminished air entry into the lungs. On the other hand, dyspnœa may be a troublesome symptom in men who have no

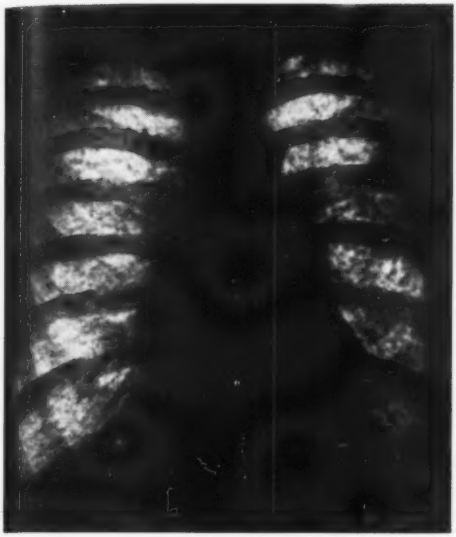

FIG. 1.—G.T., AGE 56. BOILER-SCALER FOR 30 YEARS. GENERALISED COARSE NODULATION. DIED 6 YEARS LATER OF BRONCHIAL CARCINOMA R.U.Z. HISTOLOGY SHOWED TYPICAL SILICOSIS.

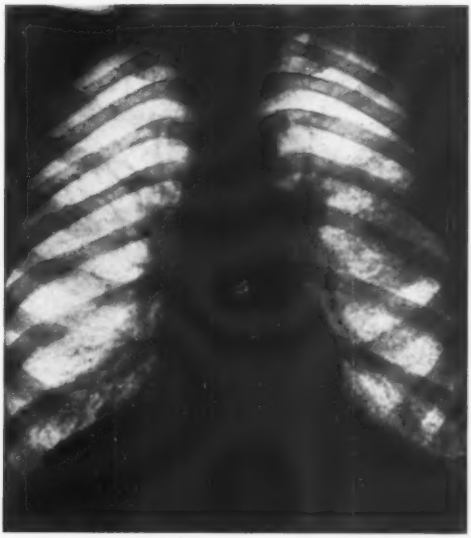

FIG. 2.—E.M., AGE 56. BOILER-SCALER FOR 40 YEARS. FINE NODULATION LOCALISED TO THE LEFT MID AND LOWER ZONES. DIED 5 YEARS LATER. P.M. SHOWED PNEUMOCONIOSIS.

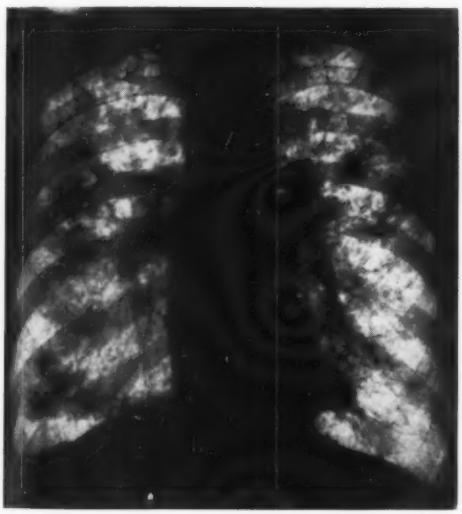

FIG. 3.—J.T., AGE 68. BOILER-SCALER FOR 20 YEARS. LEFT TRADE 22 YEARS AGO. NODULATION WITH COALESCENCE IN BOTH LUNGS. FILM TAKEN 14 YEARS PREVIOUSLY SHOWED GENERALISED NODULATION WITHOUT ANY COALESCENCE.

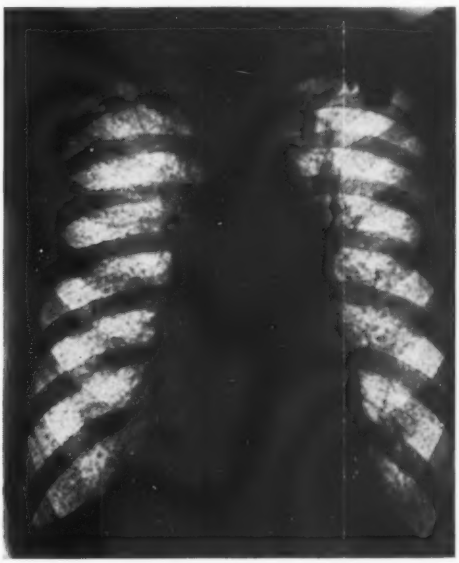

FIG. 4.—A.S., AGE 50. BOILER-SCALER FOR 33 YEARS. NODULATION OF MILIARY DISTRIBUTION. (CALCIFICATION IN L.U.Z.) FOLLOWED UP FOR 14 YEARS WITHOUT SHOWING ANY RADIOLOGICAL CHANGE.

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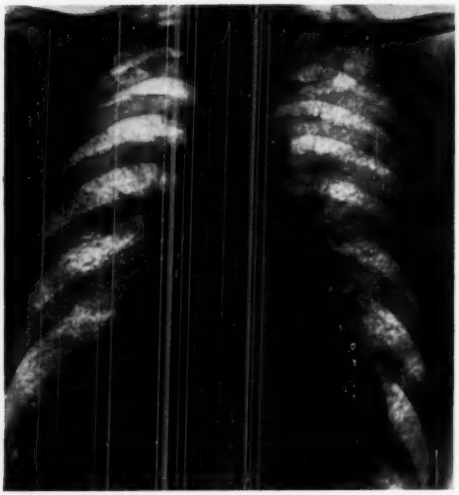

FIG. 5.—T.F., AGE 48. BOILER-SCALER. EXTREMELY FINE MILIARY NODULATION. FOLLOWED UP FOR 15 YEARS WITHOUT SHOWING RADIOLOGICAL CHANGE.

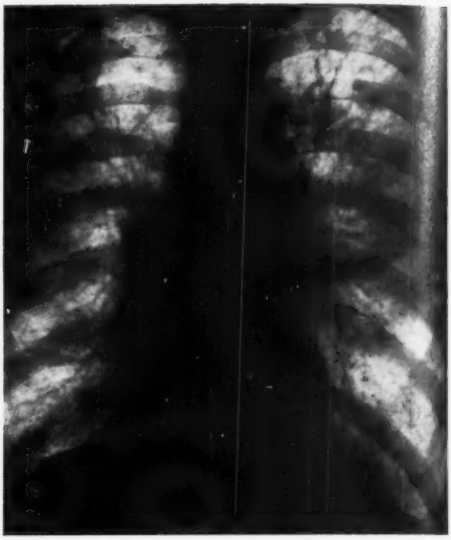

FIG. 6.—H.G., BOILER-SCALER. DIFFUSE FIBROSIS.

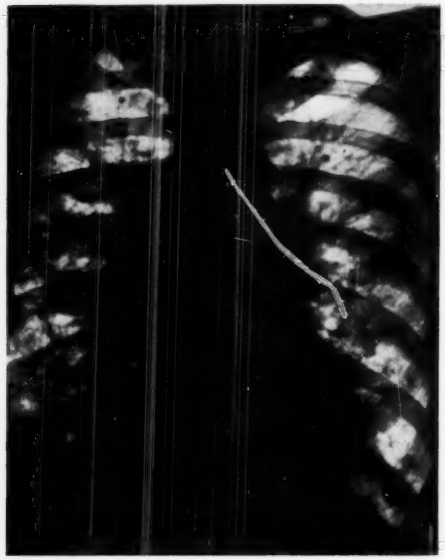

FIG. 7.—W.M., AGE 48. BOILER-SCALER 5 YEARS (28 YEARS PREVIOUSLY). FIBROSIS AND NODULATION. BRONCHIECTATIC CAVITIES IN RIGHT LUNG. SPUTUM PERSISTENTLY TB. NEGATIVE.

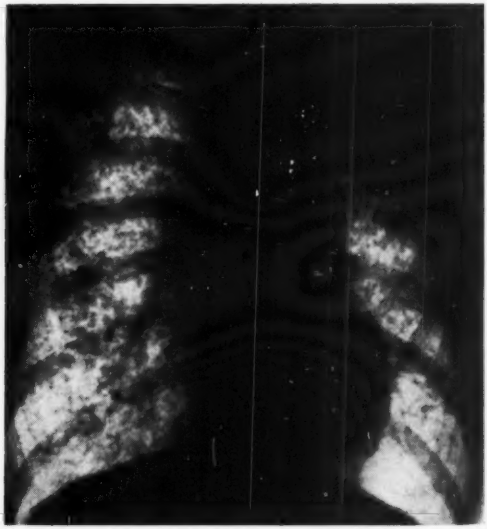

FIG. 8.—R.B., AGE 61. BOILER-SCALER FOR 45 YEARS. COARSE NODULATION, L. BASAL EMPHYSEMA. LARGE MASS LEFT UPPER ZONE. SMALLER MASS RIGHT UPPER ZONE.

To face page 45

obvious emphysema clinically and in whom radiological lesions are absent. Again, this finding has its counterpart in certain miners who exhibit a similar discrepancy between symptoms and signs

Physical Signs

The signs are no guide to the presence or extent of the lesions, as advanced pneumoconiosis may be present with few or no auscultatory signs, although there are usually basal rales indicating passive congestion of the lung or bronchiectasis. Harding *et al.* (1947) showed the presence of bronchiectatic changes in an upper lobe. Their case also had marked right ventricular cardiac hypertrophy.

Rarely, the sputum is "jet-black" (melanoptysis). In one case the chemical analysis of such sputum was as follows:

First analysis: wet 0·005 per cent., dry 0·095 per cent. silica.

Second analysis: wet 0·001 per cent., dry 0·014 per cent.

Black sputum may be expectorated many years after the cessation of boiler scaling and is not therefore derived from recently inhaled dust. It has also been shown pathologically that multiple small cavities obvious even on naked-eye inspection are present within areas of black lung. Necrosis and cavitation can therefore be postulated as the source of the black sputum.

Radiological Findings

The diagnosis of boiler scalers' disease rests essentially on the radiological findings. These appearances are not uniform, and we have met with several types.

(1) Increased lung striation. This may be (*a*) generalised and bilateral or (*b*) localised and unilateral. We are inclined to regard this type as the initial stage of the disease, but unfortunately we have not had the opportunity of doing the periodical X-rays necessary to establish this belief.

(2) Nodulation along the course of increased lung striations. This can also be either general or localised, and the size of the nodules may vary from very fine to somewhat coarse.

(3) Nodulation. We have noticed two groups: (*a*) bilateral generalised large nodules (Fig. 1), (*b*) localised small or fine nodules (Fig. 2). The opinion frequently expressed that pneumoconiosis must be symmetrical and/or bilateral is certainly incorrect. Post-mortem examination of one case (Fig. 2) whose X-ray (five years previously) revealed nodulation localised to the left lower zone showed that definite pneumoconiosis was present. Apart from this important evidence, we base our opinion on our experience of localised pneumoconiosis seen in men working in other dusty industries with known occupational hazard.

(4) Nodulation with coalescence. This requires no further elucidation and is illustrated in Fig. 3. The case is of interest because it shows the coalescence to be a later stage of an originally generalised nodulation which developed although the man was no longer working at his trade. Our impression is that coalescence is not as frequent or as extensive in boiler scalers' pneumoconiosis as in graphite workers and in miners.

(5) Miliary pattern. Radiologically this type is difficult to distinguish from miliary tuberculosis, but the difference between it and generalised nodulation is easily seen on comparison of Fig. 1 and Figs. 4 and 5. The two cases of this type were initially treated for chronic miliary tuberculosis, and the correct diagnosis was finally established by strict adherence to the old clinical maxim that chronic miliary tuberculosis should only be diagnosed by a process of exclusion. These two cases were followed up for fourteen and fifteen years respectively, the miliary pattern remained unchanged throughout and there was no evidence of coalescence.

(6) Fibrosis. The appearance of bilateral fibrosis can be recognised easily by the irregular running strands, not confined to the normal topography of the lung markings (Fig. 6). There is no difficulty in distinguishing fibrosis from the increased lung striation described in group 1. We have to report one case of unilateral fibrosis complicated by several bronchiectatic cavities with fluid levels (Fig. 7). These cavities were definitely not tuberculous, as the sputum was persistently negative for tubercle bacilli. (We have seen similar unilateral fibrosis with cavitation in a docker with pneumoconiosis due to grain dust.)

Complications

The complications which occur in boiler scalers are chiefly tuberculosis and bronchial carcinoma.

Tuberculosis

Every case of occupational lung disease must be investigated regularly for tuberculosis, which is a frequent complication. It presents itself in boiler scalers in three different ways:

(*a*) The patient, known to be a boiler scaler, shows typical X-ray evidence of tuberculosis without any manifestations to suggest pneumoconiosis. The sputum is usually positive. The lung shadows in such cases are as a rule in the upper zones and may be confined to one side, but the absence of X-ray evidence of pneumoconiosis is no justification for assuming that such lesions may not be present histologically.

(*b*) Tuberculosis becomes superadded where previous routine examinations have revealed the presence of uncomplicated pneumoconiosis.

(*c*) Radiography may suggest uncomplicated pneumoconiosis, but, surprisingly, the usual routine test shows tubercle bacilli in the sputum. In such cases it is often difficult to decide whether the lesions are due to tuberculosis and pneumoconiosis or to tuberculosis alone.

Carcinoma

In the present series four cases died of carcinoma, three of whom were bronchogenic and one intestinal with secondary carcinomatosis of the lungs. Needless to say the numbers are too small to permit of any conclusions concerning the possible carcinogenic properties of the dust, although we know of one further case of a boiler scaler with pneumoconiosis and bronchial carcinoma. A short account of these four cases may be useful, as each presents some individual feature of interest.

CASE 1. G.T. manifested nodular silicosis on a film taken at our chest clinic in 1938. Six years later he developed bronchial carcinoma in the right upper lobe. This case has been described by Harding, Tod and McLaughlin (1944) and is of special interest because of the histological evidence of definite silicosis.

CASE 2. A.R. was first seen as a case of bronchial carcinoma of the right upper lobe. Unlike case 1, there was no radiological evidence of pneumoconiosis. In spite of this sections of the lung revealed a slight degree of simple pneumoconiosis (Professor J. Gough).

CASE 3. G.D. also had no radiological evidence of pneumoconiosis. Advanced bronchial carcinoma was present in the left upper lobe, confirmed by bronchoscopy (Mr. A. M. Mair). No post mortem was done.

CASE 4. R.D. is of special interest from the differential diagnostic point of view. His film showed an extensive diffuse nodulation with dense shadows in the hilar regions, and he was shown to us as a case of silicosis in a boiler scaler. We hesitated to accept this diagnosis for two reasons: firstly, the general radiological appearance did not conform to the usual pneumoconiotic pattern, and secondly, the patient complained of excruciating pains in the chest, which may be a feature of carcinomatosis, especially with mediastinal or hilar involvement. A tentative diagnosis of carcinomatosis was made, and this was confirmed by post mortem and histologically. Professor Gough also reported: " There is very little dust and only a trivial amount of pneumoconiosis with very slight focal emphysema."

SPONTANEOUS PNEUMOTHORAX

This may not be a complication in the strict sense, but we have seen one case of concentric pneumothorax, with definite nodular lesions in both lungs, in a boiler scaler.

Discussion

We wish to confine our discussion to the radiological findings as related to the pathology. Post-mortem and histological examinations are not yet available of every group described in our section on radiology. Fig. 1 shows bilateral coarse nodulation. Harding *et al.* (1944) gave a detailed report on the pathology in this case. They found typical silicotic nodules in both lungs. On the other hand, they published a further case (1947) showing similar nodulation on X-ray, but histology revealed " a form of pneumoconiosis in which typical silicotic nodules were not found." Two of our other cases in which post-mortems were performed showed much finer nodulation than those quoted above, and one was unilateral. Professor Gough's report states: " The condition is indistinguishable from coal workers' pneumoconiosis, both in its naked eye and histological characteristics." The total silica contents of portions of the dried lungs in these two cases were determined by the method used by Fowweather, with the following results:

	Silica Percentage
1st case	0·20
2nd case	0·17

These results, although no higher than those obtained on normal lungs, do not rule out the possibility of silicosis whose diagnosis depends upon the patho-

logical rather than the chemical findings. Fowweather refers to the case of a fibrotic lung, containing scattered dense nodules, having only 0·21 per cent. of silica. (A full report on the pathological aspect of these two cases will be published by Dr. A. Massie, who performed the post-mortem examinations.) Another boiler scaler who died of bronchial carcinoma showed no radiological evidence of pneumoconiosis. In view of his occupational history, however, a post-mortem was performed and the lung sent to Professor Gough, who reported the presence of " a slight degree of simple pneumoconiosis." This case illustrates the well-known fact that pathological evidence, albeit slight, may be found in spite of the absence of X-ray manifestations. It seems, therefore, that symptoms, especially dyspnœa, in patients with a dusty occupation, should not be dismissed too readily, even if the X-ray is clear.

The literature of the pathology of boiler scalers' disease is far from complete. No post-mortem has been done on cases showing a generalised fine miliary pattern (Fig. 5). This is indeed a very interesting condition, in so far as the radiological picture has remained unchanged for fifteen years. It is possible that histological examination of such cases will reveal a pathology differing from that already described. There is no evidence of the pathological nature of the lesions which present themselves as coalescent shadows (Fig. 3) and also of the large roundish shadows (Fig. 8). Similar shadows seen in a case of a graphite worker were wrongly assumed to be solid masses. They were found on post-mortem to be cysts containing black fluid. Moreover, the pathology of the marked fibrotic strands (Fig. 6) is not known, and we can only say that we have found a similar picture in a case of a graphite worker, This type of fibrosis seems to have little in common with the usual appearance one associates with silicosis or pneumoconiosis.

The pathological evidence available so far on six cases shows that with one exception of typical silicosis all the other cases revealed pneumoconiosis.

Conclusions

It has been shown with certainty that boiler scalers are exposed to high concentrations of silica-containing dust, that their lungs suffer damage as a result, and that the lesions are due to silicosis or pneumoconiosis, as proved by histological sections. In spite of this, boiler scalers are not eligible for compensation, because their occupation is not yet expressly mentioned in the official " schedule." At the time of writing the whole problem of the method of prescribing pneumoconiosis is being reconsidered by the Industrial Injuries Advisory Council, at the request of the Minister of National Insurance. It is to be hoped that one result of their deliberations will be the addition of boiler scaling to the " schedule." When this occurs the workmen, like miners, stone masons and graphite workers, will undergo routine examinations for the purpose of detecting both pneumoconiosis and tuberculosis.

Summary

Pneumoconiosis has been shown to occur in boiler scalers in Hull.

The diagnosis rests on radiological findings, six types of which have been described. The pathological evidence so far available has been discussed in relation to the X-ray manifestations.

Complications such as tuberculosis and cancer occur as in other dusty occupations. In some of these cases very slight simple pneumoconiosis has been demonstrated histologically, even in the absence of any radiological manifestations.

We are very grateful to Professor J. Gough of Cardiff for his valuable help and reports on pathological specimens.

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THE MIDDLEBROOK-DUBOS REACTION IN RELATION TO THE CLINICAL ASPECTS OF PULMONARY TUBERCULOSIS

By K. F. W. Hinson, A. Richardson Jones and J. A. Chamberlin
From the London Chest Hospital

The adaptation of the hæmagglutination reaction to the laboratory study of tuberculosis is due to Middlebrook and Dubos (1948). Their discovery has revived and immensely stimulated research into the antigenic structure of the tubercle bacillus and the antibody response to infection. The steady growth of literature on this subject is evidence of the great interest aroused.

The reaction as originally described employed the red blood cells of the sheep which had been sensitised by extracts of tubercle bacilli or the media in which they had been cultured. They showed that agglutinins to such cells appeared in the sera of rabbits which had previously been immunised with B.C.G. and also in tuberculous patients. No agglutination in titres of 1 : 8 was given by specimens from patients with diseases other than tuberculosis or from normal individuals. In view of the high proportion of " false negatives " given by complement-fixation reactions in the diagnosis of tuberculosis (Pinner, 1925) it is significant that a pooled sample of twenty positive Wassermann sera gave only partial agglutination at a dilution of 1 in 20.

Scott and Smith (1950) demonstrated that the commercially available Old Tuberculin 4X(Lederle) was an efficient sensitising agent of sheep cells. They reported results in recent B.C.G. vaccinees and tuberculous patients. Most authors accept a titre of 1 : 8 as significant; however, it should be noted in Table 1 that Scott and Smith have recorded 1 : 2 as positive.

Again using the Lederle 4X Tuberculin, Rothbard, Dooneief and Hite (1950) applied the test to a series of tuberculous and non-tuberculous patients. Their results in the former group from which extra-pulmonary tuberculosis has been excluded are given in Table 1. No agglutination was given by 110 normal individuals or a series of 81 patients with non-tuberculous pulmonary conditions, nor yet in a further series of 25 with syphilis.

The necessity for the preliminary absorption of heterophile or anti-Forssman agglutinins against unsensitised sheep cells was obviated by Middlebrook (1950a), who used the patient's own washed corpuscles for sensitisation by the concentrated tuberculin. A titre of 1 : 16 was recorded as positive and twenty-four sera from tuberculous cases gave agglutinations at or above this level. Results from ten non-tuberculous controls, six of whom, however, might be expected to have raised serum globulin, were all negative. In this paper Middlebrook described a modification of the test. If suitably absorbed guinea-pig complement was added to the system, hæmolysis of the cells might occur. As there was no obvious correlation between the titres in the two reactions with any one serum, it was suggested that different antibodies may be involved.

Hilson and Elek (1951) used human Group O rhesus-negative cells which

were sensitised by various extracts of tuberculin preparations, including P.P.D. They employed the Chown capillary tube technique to demonstrate the hæmagglutinins. Their results, which are summarised in Table 1, confirm the findings of Middlebrook and Dubos that a crude polysaccharide fraction is the most efficient sensitising agent.

It was claimed by Thalhimer and Rowe (1951) that the tests may be performed more quickly if they are set up on microscope slides rather than by the tube method of previous investigators. They also suggest that this modification may be more sensitive.

Laboratory Techniques

Fresh human Group O rhesus-negative erythrocytes were thrice washed in isotonic saline and resuspended in a 1 in 10 dilution of Lederle 4X Old Tuberculin in the ratio of one volume of packed cells to ten volumes of tuberculin solution. The mixture was incubated for one hour with frequent shaking. At the end of this time the erythrocytes were again washed three times in saline and made up to a final suspension concentration of 1 per cent. for use in the reaction.

The serum for examination was separated from the clot as soon as it was received in the laboratory and inactivated by heating to 56°C. for half an hour: thereafter the serum was stored at −20°C.

The reaction proper was performed by preparing doubling dilutions of the serum in 7×50 mm. tubes, the dilution range being 1 : 2 to 1 : 32. One volume of tuberculin-treated cells was added to each of the tubes, which were thoroughly shaken and then incubated for at least two hours at 37°C. Reading was carried out by observing the sedimentation pattern of the cells with the aid of a magnifying mirror.

The highest dilution at which irregularity of sedimentation pattern was observed was taken as the end-point. No attempt was made to titrate out to its limit a serum which agglutinated the cells at 1 : 32. The justification for this will be apparent from the results given below.

In addition to this procedure all sera were subjected to a further test. This consisted of the performance of a Coombs' antiglobulin reaction (Coombs, Mourant and Race, 1945) on tuberculin-treated cells after they had been allowed to react with a 1 : 2 dilution of tuberculous serum for at least one hour. The cell suspension used was approximately 2 per cent. After removal of the supernatant 1 : 2 serum, the cells were washed three times with saline and one drop of suitably diluted anti-human rabbit serum added to the packed cells from the final washing. After thorough shaking, the tubes were allowed to stand at room temperature for ten minutes; they were then reshaken, the suspension transferred to a glass slide and observed for macroscopic agglutinates while the slide was gently rocked. Non-appearance of agglutinates after two minutes was taken to indicate a negative result. The results were usually very clear-cut, massive agglutination occurring in a few seconds in the case of positives.

It is not proposed to include in the present paper the results of this latter reaction, as the authors feel that the major issues would thereby be unduly complicated. It suffices to say that the findings provide strong evidence for

the existence of at least one other antibody of different specificity from that responsible for the classical hæmagglutination reaction. Further work remains to be done, the results of which will be reported elsewhere.

Results

The overall picture of our results is summarised in Diagram 1. Table 1 gives a comparison of the results obtained in this series, together with those of previous workers in this field with respect to tuberculous sera and non-tuberculous controls.

The material used in this series to provide non-tuberculous controls may be divided into two groups:

1. Twenty apparently healthy adult blood donors on the panel of the North London Blood Supply Depot.
2. Forty-four non-tuberculous intrathoracic disease cases admitted to the London Chest Hospital.
   Tuberculosis was excluded clinically, radiologically and by repeated sputum examinations. Frequently pneumonectomy and lobectomy specimens from these patients allowed active tuberculosis to be excluded on histological grounds.

The cases of known pulmonary tuberculosis consisted of an unselected series of 130 from the London Chest Hospital Country Branch, Arlesey. They represented all phases in the natural history of the disease, from newly discovered minimal lesions to advanced chronic disease. The age of the patients ranged from 14 to 51, the average being 30 years. The duration of their disease since the time of first diagnosis was recorded, the average being 3·3 years. The extent of the pulmonary lesions was assessed according to radiological appearances, following the classification of the National Tuberculosis Association of America (1940) as cited by Heaf and Rusby (1948).

Minimal.—Slight lesions without demonstrable excavation confined to a small part of one or both lungs. The total extent of the lesion, regardless of distribution, shall not exceed the equivalent of the volume of lung tissue which lies above the second chondrosternal junction and the spine of the fourth or body of the fifth thoracic vertebra on one side.

Moderately Advanced.—One or both lungs may be involved, but the total extent of the lesion shall not exceed the following limits:

Single disseminated lesions which may extend through not more than the volume of one lung, or the equivalent of this in both lungs.

Dense and confluent lesions which may extend through not more than the equivalent of one-third the volume of one lung.

Any graduation within the above limits.

Total diameter of cavities, if present, estimated not to exceed 4 cm.

Far Advanced.—Lesions more extensive than moderately advanced.

The proportions in these groups were: minimal, 36 per cent.; moderately advanced, 39 per cent. ; far advanced, 25 per cent.

For the purposes of Diagram III, these will be referred to as Classes 1, 2 and 3.

A number of the cases had been admitted with a view to major surgery, and had received previous sanatorium and domiciliary treatment. An analysis of treatment previous to the investigation showed that 69 per cent. of the cases

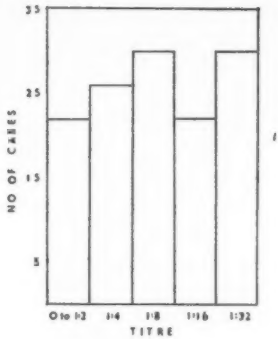

DIAGRAM I

The distribution of 130 unselected cases of pulmonary tuberculosis according to hæmagglutination titre.

TABLE I.

Author	*Tuberculous Cases tested*	*Percentage Positive*	*Sensitising Agent*	*Cells*	*Technique*	*Remarks*	*Non-tuberculous Control*	*Percentage Positive*
Middlebrook and Dubos	6	100	H37RV Extracts	Sheep	Tubes	1 control was a pool of 20 W.R.+sera	11	0
Scott and Smith	56	76	Lederle 4X	Sheep	Tubes	Titre of 1 : 2 " positive "		
Rothbard *et al.*	160	92	Lederle 4X	Sheep	Tubes		226	0
Smith and Scott	104	80	Lederle 4X	Sheep	Tubes	Titre of 1 : 2 " positive "	132	30
Middlebrook	27	90	Lederle 4X	Sheep	Tubes	Titre of 1 : 16 " positive "	10	0
	27	90	Lederle 4X	Sheep	Tubes	Hæmolytic modification	10	0
Sohier *et al.*	20	55	Tuberculin Precipitée	Sheep	Tubes		240	4
Sohier *et al.*	31	74	I.P. 48	Human	Tubes		121	6
Gernez-Rieux and Tacquet	185	85	I.P. 48	Sheep	Tubes		185	7
Hilson and Elek	66	67	O.T.	Human	Chown	Titre of 1 : 2 " positive "	89	3
	66	92	Polysaccharide Fraction	Human	Chown	Titre of 1 : 2 " positive "	60	0
Thalhimer and Rowe	75	70	Lederle 4X	Sheep	Tubes	Sera from 57 patients	31	40
	75	96	Lederle 4X	Sheep	Slides	Sera from 57 patients	31	30
Kirby *et al.*	42	69	Lederle 4X	Sheep	Tubes	Results in pulmonary tuberculosis only	251	10
Present Investigations	130	63	Lederle 4X	Human	Tubes		64	5

had undergone more than six months' bed rest or sanatorium régime, and 70 per cent. had past or present collapse measures. Only 10 per cent. had no chemotherapy or antibiotics; 78 per cent. had received or were still receiving streptomycin and P.A.S., and 12 per cent. had received P.A.S. alone.

No cases in their first post-operative month were included in the series. It is claimed that this series is a selected one only in so far as the cases had been considered suitable for hospital treatment (and untreatable cases are forming a progressively smaller proportion of the tuberculous population).

Toxæmia was recorded as present in those cases which showed one or more of the following features, without obvious cause other than pulmonary tuberculosis: pyrexia, persistently raised pulse rate, continued loss of weight, marked constitutional symptoms or a raised E.S.R.

The E.S.R. was measured at the same time as blood was collected for the serological tests, using Westergren's method.

The progress of each case was assessed by reference to the serial X-rays, sputum conversion and the changes in the signs of toxæmia. The cases were divided broadly into "Static," "Improving" or "Deteriorating."

Effect of Streptomycin

Table II summarises the effect of streptomycin therapy on the incidence of positive reactions. It will be seen that there is no difference in the incidence of positive reactions between those patients who have never received streptomycin and those who have completed the course of therapy. Of the patients who were receiving streptomycin at the time blood was taken for the test, only 48 per cent. showed positive reactions. This would appear to extend to humans the work of Gernez-Rieux and Tacquet (1950a), who showed a similar phenomenon in rabbits. However, if the data within the double lines in Table II are treated as a fourfold table (Fisher, 1950) by the application of the equation:

$$X^2 = \frac{(ad-bc)^2\ (a+b+c+d)}{(a+b)\ (c+d)\ (a+c)\ (b+d)}$$

and the table of X^2 entered with $n+1$, then P is found to be greater than 0·20, which denies significance to this difference.

Table II.

Streptomycin Course	*Over* 1 : 8	*Under* 1 : 8	*Total*	*Percentage over* 1 : 8
Had	53	27	80	66
Not had	19	10	29	66
Having	10	11	21	48

Analysis of reactions in patients at different stages of streptomycin therapy. The figures within the heavy lines constitute a fourfold Table.

Effect of E.S.R. and Duration of Disease

Hilson and Elek (1951) published "scattergrams" of titre against E.S.R. (Westergren) and of titre against duration of disease. They failed to show

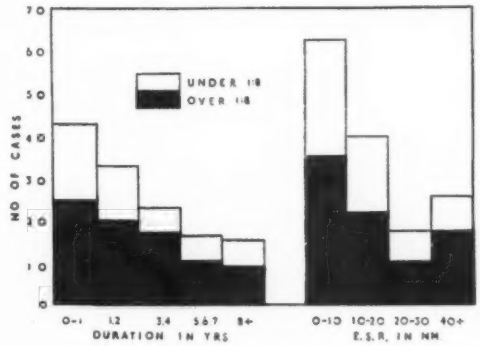

Diagram II

To illustrate the lack of correlation between duration of disease and agglutinin titre (left-hand side) and E.S.R. and agglutinin titre (right-hand side).

correlation with respect to either factor. Diagram II gives good confirmation of their findings.

Effect of Extent

The work of Kirby *et al.* (1951) suggested that some correlation might be found between the anatomical extent of pulmonary tuberculosis and the result

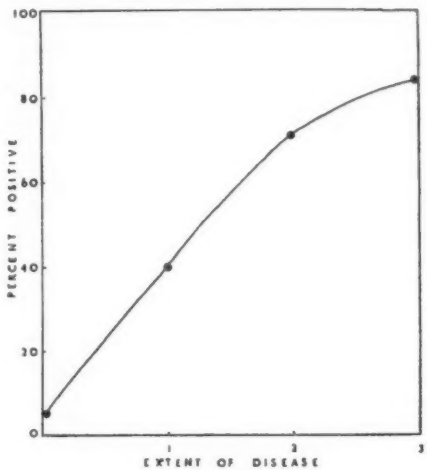

Diagram III

To illustrate the proportionate increase of positive hæmagglutination reactions with increasing anatomical extent of disease.

(1) Minimal.
(2) Moderately Advanced.
(3) Far Advanced.

of the hæmagglutination reaction. Diagram III presents strong evidence to support this. The relation between extent and the likelihood of finding a positive reaction seems to be almost a linear one. It should be borne in mind, however, that our series includes no cases of terminal disease. Smith and Scott have shown such subjects to give a very low incidence of positive reactions.

Effect of Age

The relationship between age and hæmagglutination is illustrated in Diagram IV. The maximum incidence of positive reactions is found in the 20-30 age group. Sorting the clinical data failed to show that this age group

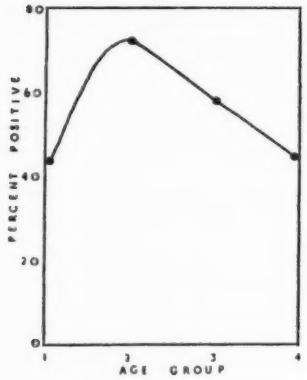

Diagram IV

The effect of age on the incidence of positive reactions. The age groups are as follows: (1) Under 20 years. (2) 20 to 29 years. (3) 30 to 39 years. (4) 40 years and over.

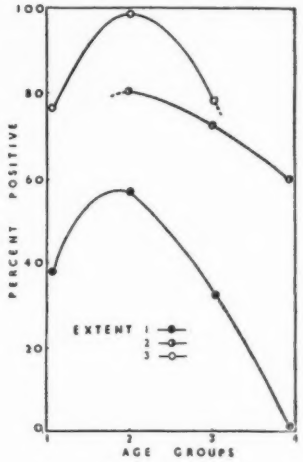

Diagram V

The independence of the effects of age and extent of disease on the incidence of positive reactions. Definition of age groups as in Diagram IV.

was weighted by an unusually high proportion of extensive disease. The independence of age and extent are well shown in Diagram V, where the most striking features are complete absence of response to minimal lesions in the over-40s and 100 per cent. response to extensive disease in the 20-30 group.

The Prognostic Value

Without serial titration of numbers of sera over a prolonged period of time it is difficult to pronounce on this aspect. A study of this type is in progress

Table III.

Group	*Number*	*Number Positive*	*Percentage Positive*
Static	31	20	66
Improving ..	85	52	62
Deteriorating ..	14	10	72

and will be reported in due course. However, a simple segregation of cases into " static," " improvers " and " non-improvers " fails to demonstrate any difference in the distribution of positives between the two groups (Table III).

Effect of Clinical type

An attempt was made to group the cases according to the predominant feature of the disease process. Those features which could be deduced from a study of the serial X-rays were considered, bearing in mind that the phases of destruction and repair always exist side by side in varying degrees. The features were: pleural effusion, cavitation, fibrotic disease, single solid foci, episodes of spread, surgically extirpated disease.

The data were sorted in many different ways in an effort to discover the existence of a possible relationship to the proportion of positive reactions.

Of all these factors, taken either singly or in combination, only the first and last give significant differences from the mean, and these cases would obviously fall into the " minimal extent " category.

The presence of toxæmia also appeared to be quite unrelated to the proportion of positive reactions.

Discussion

Ideally, a serological reaction in tuberculosis should be specific and give a high proportion of positive results in all stages of such a chronic disease, and produce quantitative indications of the activity of the infection. However, our investigations would appear to confirm the previous reports that the hæmagglutination reaction in its present form has no value in the diagnosis or prognosis of pulmonary tuberculosis.

It cannot be regarded as sufficiently specific, as 5 per cent. of the non-tuberculous controls were positive and many negative results were obtained from patients in various stages of the disease. Nevertheless, the series does show a higher proportion of positive results with increasing anatomical extent of disease. This is in agreement with Kirby *et al.* and contrary to the findings of Rothbard *et al.*, who demonstrated only small differences in " minimal, moderately advanced and far advanced " pulmonary cases.

We have been unable to find an analysis of results into age groups to compare with Diagram IV, which would suggest that the test might most usefully be employed in the 20-30 age group. It would be unlikely to confirm the diagnosis of minimal disease in patients over 40 years of age.

Recent Mantoux tests have not been performed on all our cases, but from the literature it is clear that the hæmagglutination reaction does not always give results in parallel with the tuberculin sensitivity. For instance, Scott and Smith, working to a titre of 1 : 2, record the variations in the two tests in human volunteers receiving B.C.G. vaccinations.

In agreement with Hilson and Elek, we were unable to display any correlation between the duration of the disease and the proportion of positive results.

If the antibodies responsible for hæmagglutination were protective they would be expected to be present in a large proportion of the cases who have maintained an equilibrium with their disease over a long period, and conversely to be present in a smaller proportion of those who have demonstrated low

resistance by extensive involvement. Our results do not confirm this thesis and suggest that the antibodies responsible for hæmagglutination are in no way protective or concerned with resistance to the infection.

Middlebrook (1950b) suggests that the hæmagglutinins were formed in response to the products of lysis of the bacilli. The increased proportion of positive results in extensive disease would be the expected finding if this were the case. The present series suggests that hæmagglutins reach significant levels only in those conditions in which the bacilli may be assumed to be multiplying. Positive results have been obtained following recent B.C.G. vaccinations (Smith and Scott, 1950; Gernez-Rieuz and Tacquet, 1950), and two of the Hilson and Elek control group who gave high titres were later found to be tuberculous. Conversely, Rothbard *et al.* had no positive findings in a group of patients whose disease was considered to be healed, and in those of the present series who had been treated by excision the test was negative. Again the process of bacterial multiplication would be retarded during the course of streptomycin therapy, and in our series the proportion of positives was lower in this group. Although the influence of lytic products cannot be excluded, it would seem probable that one or more of the metabolic products of proliferating bacilli must also be acting as an antigenic stimulus.

In their original paper Middlebrook and Dubos showed, on the basis of experiments involving the inhibition of hæmagglutinins by various specific tuberculin fractions, that the most efficient sensitising agent of the red blood cells was a polysaccharide complex. Working with a "crude carbohydrate fraction" which they had prepared, Hilson and Elek confirmed this. Polysaccharide is not, however, the only fraction adsorbed on to the red cells, as De Witt and Birkeland (1951) report that tuberculin-sensitised cells or their stroma may be used to demonstrate tuberculin sensitivity in infected animals, a property usually ascribed to the protein fraction. However, it should be noted that P.P.D. may contain up to 3 per cent. of polysaccharide (Siebert and Dufour, 1948).

The *in vitro* inhibition experiments were cited by Middlebrook (1950b) as evidence that in gravely ill patients the antigenic substance may be present in such quantities as to react with all the antibody formed, so that there is insufficient of the latter to agglutinate the sensitised cells. Certainly such negative results are common with sera from patients in the terminal stages. It was thought that cross-reaction experiments between such sera and high titre positive sera might determine whether the negative results are due to the depression of antibody formation or to the "swamping" effect of excessive antigen. However, preliminary trials of this method during the present investigations would suggest that the former explanation is the more probable.*

Summary

1. The literature has been reviewed and summarised.
2. The results of the hæmagglutination reaction in 130 unselected hospital cases of pulmonary tuberculosis are given. The proportion of positive results increases with the extent of disease, and is influenced by age.

* A.R.J. unpublished data.

3. The conventional test has no value in the diagnosis or prognosis of this disease.
4. A new technique for the demonstration of tuberculin antibodies has been described.

We wish to thank the staff of the London Chest Hospital for permission to investigate and discuss their cases, and also the director of the North London Blood Supply Depot. The tuberculin used was kindly supplied by Cyanamid Products Ltd.

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KRUKENBERG TUMOUR PRESENTING AS PLEURAL EFFUSION

REPORT OF A CASE SIMULATING MEIGS' SYNDROME

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Sero-fibrinous pleurisy—pleurisy with effusion—may be associated with an obvious underlying lung lesion or the effusion may be the only apparent abnormality, as with the so-called "idiopathic pleural effusion." This in a young adult, especially when the fluid is a lymphocytic exudate, is of tuberculous origin unless proved otherwise.

The principal causes of transudates are heart failure, hydræmic nephritis and new growths.

Other rarer causes of effusions include certain pneumonias, eosinophilic lung infiltrations, Meigs' syndrome, etc.

The case about to be described, a young female of 22, had initially a unilateral lymphocytic exudate and later developed a bilateral pleural effusion and ascites consequent upon a malignant ovarian tumour, in contradistinction to the more usual benign ovarian fibroma of Meigs' syndrome.

Case Report

M. B. Female æt. 22, an identical twin who had previously been in good health, was referred to one of us on 24.1.51 with a history of pleuritic pain in the left chest that had started a fortnight previously and was followed by increasing dyspnœa. There were no constitutional symptoms, but she had a slight unproductive cough over the same period.

The previous medical history was not relevant and there was no known case of tuberculosis in the family, which was confirmed later by X-ray examination of the contacts.

On examination she appeared unwell, pale and dyspnœic; the upper respiratory tract was normal and there were no palpable lymphatic glands. Her weight was 8 stone 1½ lb., temperature 96·8° F. and pulse 104. Clinical examination of her chest revealed a large left pleural effusion; nothing abnormal was noted in the abdomen or in the central nervous system.

X-ray examination of her chest confirmed the presence of a large left pleural effusion; on the right side the transverse fissure was thickened, and there was an exaggeration of the lung markings in the middle and lower zones somewhat suggestive of miliary mottling.

A laryngeal swab was negative for acid-fast bacilli on direct examination, and subsequent culture did not reveal tubercle bacilli. The B.S.R. was 30 mm./hour (Westergren).

A paracentesis of the chest was performed, 400 c.c. of clear, straw-coloured fluid being removed and later reported on as follows: "Direct film shows polymorphs and mononuclear cells. Culture—sterile. Film stained by Ziehl-Neelson: no T.B. seen."

She was advised to rest in bed at home, but when a fortnight later it was

found that she had lost nearly a stone in weight and the effusion had increased, she was admitted to the Plaistow Hospital Chest Unit under the care of one of us (P. E.) on 13.2.51. Here she was kept on bed rest, and although a low-grade, irregular pyrexia persisted, the position appeared to remain stabilised in so far as there was little alteration in the clinical and radiological appearances. She improved appreciably and gained 3 lb. in weight and the B.S.R. fell from 72 mm. to 56 mm./hour. Mantoux tests done at this time were negative to 1/100, and repeated examinations of laryngeal swabs and gastric washings were negative for tubercle bacilli on culture. The pleural fluid remained clear, contained moderate numbers of lymphocytes and epithelial cells, and no tubercle bacilli or other organisms were found on direct examination, culture or guinea-pig inoculation.

She was transferred to the Pleural Effusion Unit, Sidcup, under the care of Dr. P. Forgacs on 6.3.51, with a presumptive diagnosis of tuberculous pleural effusion.

There were no symptoms on admission, but shortly afterwards a small left supraclavicular lymph gland was discovered and it was noticed that there was some distension of the abdomen below the umbilicus. Palpation of the abdomen after the bladder was emptied revealed no abnormality and the swelling was attributed to a full bladder.

For the next six weeks there were apparently no striking clinical grounds to suppose that the case was other than of tuberculous etiology. Two aspirations of pleural fluid (11 oz. and 5 oz.) were reported on as " Dark, amber fluid. Lymphocytes 85 per cent. Negative for A.F.B." The Mantoux tests were repeated and again found negative to 1/100.

On 26.4.51 the patient suddenly became acutely ill with dyspnœa, cyanosis and tachycardia from 100-150 per minute. Physical examination, later confirmed by X-ray, revealed the presence of a massive right pleural effusion. Aspiration of 2 pints of fluid afforded no relief, and a course of streptomycin (0·5 gm. I.M. b.d.) and P.A.S. (18 gm. daily in divided doses) was initiated, as it was felt that, in spite of the absence of pyrexia, an atypical miliary spread might have occurred.

In order to relieve dyspnœa a total of 8 pints of fluid were removed from the right chest and ¼ pint from the left chest during the next three weeks. The early samples showed 25 per cent. endothelial cells and no eosinophiles, and the later samples 22 per cent. eosinophiles and no endothelial cells.

At this time the presence of a lower abdominal tumour was definitely established and the diagnosis of Meigs' syndrome was considered. In consultation Mr. L. P. Clark found the right ovary greatly enlarged and advised laparotomy. Questioning now elicited the information that her periods, which had previously been normal, were scanty on the last two occasions and had lasted only two days instead of five.

At operation on 15.5.51 a large right ovary showing cystic areas on the surface was removed. The left ovary was slightly enlarged and showed the same cystic appearances, but was not removed.

The post-operative course was uneventful and for three weeks she improved; the cyanosis disappeared, the pulse rate fell to 90-100 per minute, and the dyspnœa was relieved to the extent that further aspirations were not necessary. On 6.6.51, however, her condition began to deteriorate; cyanosis, tachycardia and dyspnœa returned and 12 ounces of fluid were removed from the left chest. No signs of a pericardial effusion were found to account for the cardiac embarrassment. Her condition steadily deteriorated and death occurred on 14.6.51, preceded for two days by œdema of the ankles.

Pathological Reports

Section of the Right Ovary Removed at Operation

This was referred to Professor R. A. Willis, University of Leeds, who reported:

"In spite of the youth of the patient I feel sure that this is a Krukenberg tumour, almost certainly secondary to carcinoma of the stomach. The structure, with groups of signet-ring cells containing mucin or pre-mucin, and the œdematous hyperplastic state of the ovarian stroma are quite characteristic. I have no doubt that her pleural effusion is due to secondary growth in the lung and pleura, and I note with interest that a large left supraclavicular gland is present (Troisier's sign). The photograph of the specimen in itself suggests the possibility of a Krukenberg tumour. In these cases the primary gastric tumour is often symptomatically quiet and may be quite small and undetectable clinically. However, it should certainly be looked for by X-ray following opaque meal, the findings of which, if positive, will clinch the diagnosis. The surgeon observed that the other ovary also was diseased, and this, too, is a common finding with secondary growths of this kind."

Summary of Autopsy Findings (Dr. C. H. R. Knowles, Southern Group Laboratory)

Slightly enlarged lymph nodes were found in the left supraclavicular and both deep cervical regions, along the greater curvature of the stomach and around the head of the pancreas. The cœliac, mesenteric and other groups appeared normal.

The visceral and parietal *pleuræ* over the whole right lung and the upper lobe and diaphragmatic surface of the left lung were densely bound together by soft pinkish gelatinous tissue. The lower part of the left pleural cavity was loculated by thickened pleura and contained a considerable quantity of yellowish fluid with some air. There were traces of interstitial emphysema in the superior mediastinum.

The *lungs* were moderately contracted and rather firm in consistency. Cut surfaces showed a uniform, fine diffuse thickening of the connective-tissue framework. No areas of bronchopneumonia, recognisable tumour masses, or other focal lesions were found.

The *pericardium* contained about ½ litre of slightly opalescent yellow fluid under tension, but the serous surfaces appeared healthy. Apart from slight generalised dilatation the *heart* was normal.

The *peritoneum* contained a moderate amount of yellow fluid, but no metastatic deposits were found.

The *stomach* was sliced at half-centimetre intervals, but was macroscopically free from growth or other abnormality.

The *uterus and Fallopian tubes* were normal and the right ovary was absent. The *left ovary* was replaced by an ovoid tumour, about 6 cm. in main diameter, with a slightly nodular surface. Its cut surface was rather soft, moist, glistening and semi-translucent, and showed minute areas of cystic degeneration; this appearance was identical with that of the right ovarian tumour removed at operation.

Nothing abnormal of note was found in any of the other organs.

Histological Findings

The *left tonsil* showed reactive hyperplasia of the lymph-nodal tissue with purulent debris in some of the crypts. A few small groups of signet-ring cells were found in the lymphatic capillaries at the periphery. A *left deep cervical node* showed no evidence of secondary deposits.

Several sections from the *lungs* were examined and the appearances in all were similar. Almost all the interstitial lymphatic vessels were distended by carcinoma cells, mostly typical signet-ring cells arranged in compact masses, but in a few areas columnar-celled acinar structures were found. Small groups of malignant cells were seen in many small veins and arteries, in some of which thrombosis and organisation had occurred. Many small arteries and arterioles showed conspicuous proliferative endarteritis which in some cases had proceeded to complete occlusion of the lumen. The lung parenchyma showed patchy collapse, congestion and œdema, and in some areas the alveoli contained a few macrophages. Only in one or two minute areas were tumour cells found within the alveoli, and no significant destruction of lung tissue was demonstrated. The *pleura* was thickened by œdematous fibrous tissue sparsely infiltrated by lymphocytes, histiocytes and occasional small groups of signet-ring cells. No tumour masses exceeding 0·2 cm. in diameter were seen in the lungs, and the condition appeared to be essentially a diffuse carcinomatous infiltration of the interstitial lymphatic network.

Sections of the *diaphragm* showed marked œdema and congestion, and there were many dilated lymphatic vessels containing differentiated adenocarcinomatous elements and small compact masses of signet-ring cells.

Sections were examined from two representative areas of the *stomach* and three of the pyloric canal. The mucosa showed early autolytic changes, but no other histological abnormalities.

A few signet-ring cells were found in the peripheral sinuses of a *pyloric lymph node*.

The *liver* showed marked chronic venous congestion which varied in extent in different areas; in some parts centrilobular necrosis with polymorphonuclear infiltration had occurred, but no evidence of secondary deposits was found.

In the *left ovary* the appearances were essentially the same as those of the right ovary removed at operation.

No noteworthy abnormality was found in sections from the myocardium, kidneys, suprarenals and cerebral hemisphere.

Discussion

Meigs and Cass (1937) and Meigs (1939) described a syndrome in which bilateral pleural effusions and ascites are associated with an ovarian fibroma, the removal of which results in recovery. As pointed out by Nelson and Dennison (1951), it would appear valid to include in the syndrome cases with pelvic tumours other than ovarian fibroma, even if they are malignant, provided there is no evidence that the accumulations of fluid are due to metastases in the lungs, pleuræ or peritoneum. The mechanism causing the serous transudates in these two groups would appear to be the same, and cases have been described in which removal of malignant tumours of the ovary

has led to reabsorption of the fluid and recovery. Schenck and Benjamin (1939) described such a case with papillary cystadenocarcinoma of the ovary, and Rubin *et al.* (1944) recorded one with malignant cystadenoma. Beresford and Aidin (1950) describe a case of Meigs' syndrome, which came to autopsy, in which the ovarian tumour proved to be an adenocarcinoma and in which gross and microscopic examination of the lungs, pleuræ and peritoneum failed to reveal metastases.

The case now described does not conform even to this wider concept of Meigs' syndrome, as the pleural effusions and possibly the ascites were due to lymphatic metastases which were widespread and extended even as far as the tonsils. The freedom of the liver from secondary deposits is remarkable, and according to Willis (1948) this is a frequent finding with Krukenberg tumours.

The interest of the case lies in the diagnostic problem in the first eleven weeks of the illness and the failure to demonstrate a primary growth at autopsy.

The importance of establishing the etiology in cases of pleural effusion is well shown in this case. A careful and complete physical examination should always be made and tubercle bacilli should be searched for vigorously by all the recognised methods, as until their presence is demonstrated in the pleural fluid or bronchial secretions the diagnosis of tuberculous pleural effusion is really only presumptive. Factors which would strengthen such a diagnosis are a family history of tuberculosis, a positive Mantoux reaction, and clinical and radiological evidence of tuberculous disease in the lungs or elsewhere in the body. The physical properties and cytology of the pleural fluid besides the bacteriology should always be determined and are of fundamental importance. In Meigs' syndrome it is a sterile transudate of low specific gravity (Schaffner and Kirkpatrick, 1946).

In the case now reported there was no evidence of tuberculosis in the family, the Mantoux tests were negative to 1/100, and repeated examinations of laryngeal swabs, gastric washings and pleural fluid failed to demonstrate tubercle bacilli. There was thus evidence against a diagnosis of tuberculous pleural effusion which was mainly supported by the early clinical course and the appearance and cytology of the pleural fluid in the early specimens.

In retrospect, the earliest clue of the true diagnosis was the " atypical " X-ray appearance of miliary mottling in the lower half of the right lung field, which was attributed to tuberculosis and ultimately proved to be due to lymphangitis carcinomatosa. We have, however, of recent years seen several cases of primary lung tuberculosis followed by a pleural effusion and ending subsequently in miliary tuberculosis. The Mantoux test in such a case may be negative and the radiological appearances may be strikingly similar (Fig. 4).

The subsequent appearance of an effusion on the other side and the finding of an ovarian tumour was very suggestive of Meigs' syndrome, but it was only the pathological examination of the right ovary removed at laparotomy that established the malignant nature of the condition. The later specimens of fluid removed from the right chest were amber-coloured and contained eosinophiles and endothelial cells in varying proportions, and the absence of lymphocytes is noteworthy.

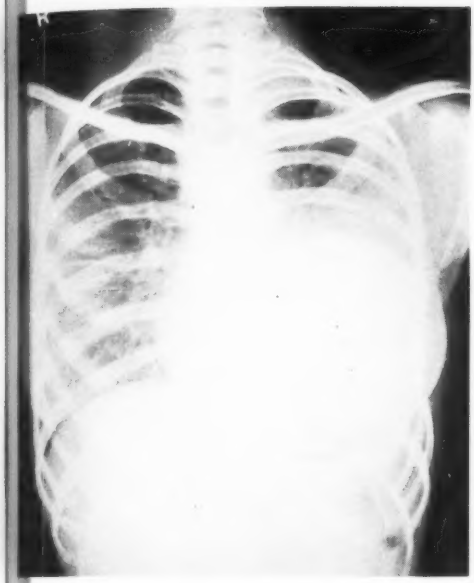

FIG. 1.—AN EARLY PICTURE SHOWING THE LEFT PLEURAL EFFUSION AND THE MILIARY APPEARANCE IN THE LOWER HALF OF THE RIGHT LUNG FIELD. THE TRANSVERSE FISSURE IS THICKENED.

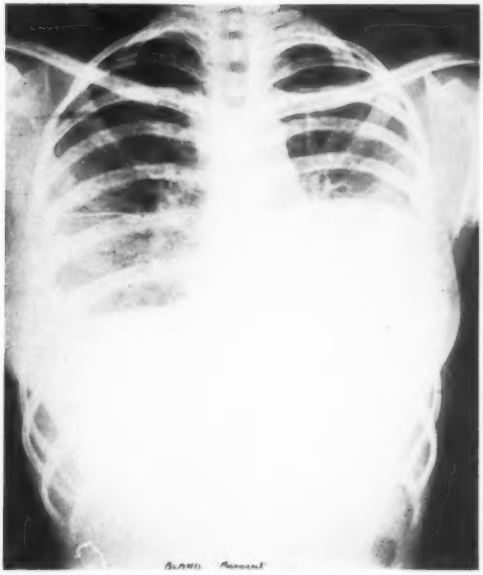

FIG. 2.—AFTER SIX WEEKS THE LEFT EFFUSION APPEARS TO BE GETTING LESS. THE RIGHT LUNG IS LITTLE ALTERED.

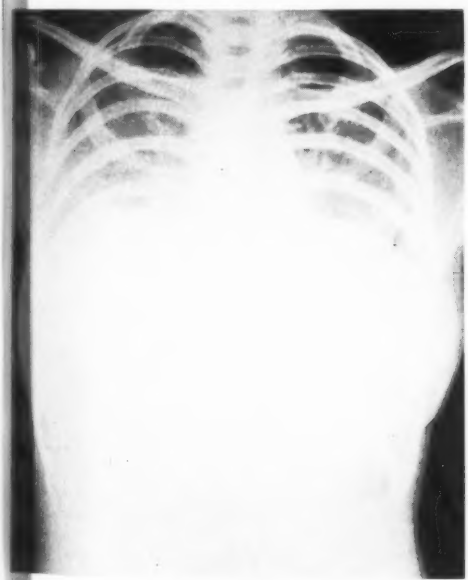

FIG. 3.—NOW A MASSIVE RIGHT EFFUSION HAS APPEARED AND MASKS THE INFILTRATION AT THE RIGHT BASE AND THE THICKENED TRANSVERSE FISSURE. THE LEFT EFFUSION IS FURTHER REDUCED FOLLOWING ASPIRATION AND AN AIR-CONTAINING POCKET CAN BE SEEN AT THE BASE.

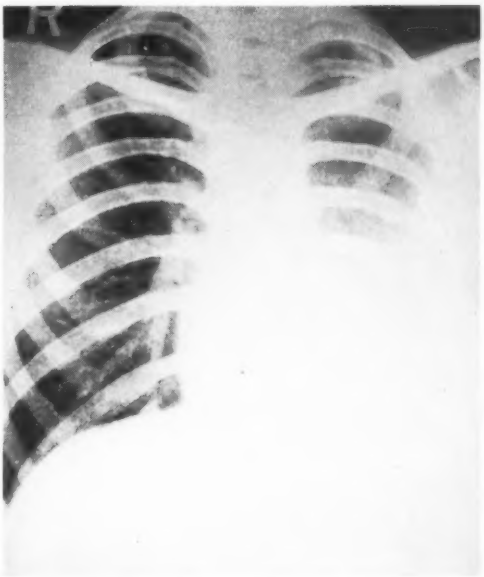

FIG. 4.—FOR PURPOSES OF COMPARISON. SKIAGRAM OF A MALE, ÆT. 17, WITH LEFT PLEURAL EFFUSION AND MILIARY MOTTLING IN ALL LUNG FIELDS. THE APPEARANCES ARE SOMEWHAT SIMILAR TO THE CASE NOW DESCRIBED.

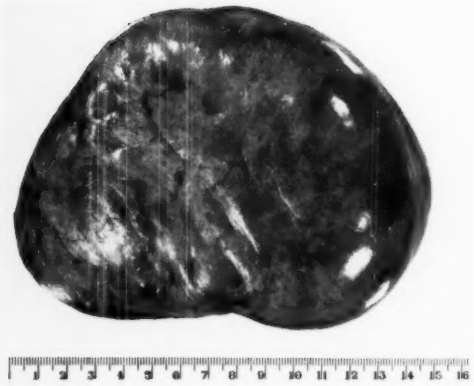

FIG. 5.—SURFACE OF RIGHT OVARY REMOVED AT OPERATION.

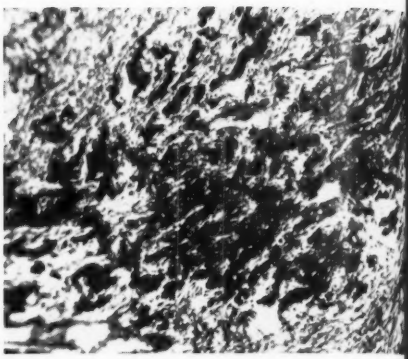

FIG. 7.—SECTION OF LUNG SHOWING DISTENSION OF THE LYMPHATIC VESSELS BY MALIGNANT SIGNET-RING CELLS IN COMPACT MASSES. THE LUNG PARENCHYMA SHOWS PATCHY COLLAPSE CONGESTION AND ŒDEMA.

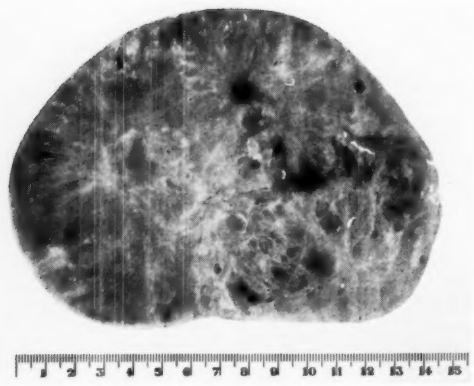

FIG. 6.—CUT SURFACE OF RIGHT OVARY REMOVED AT OPERATION.

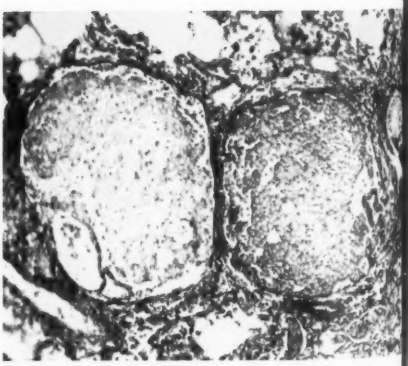

FIG. 8.—SECTION OF OVARY SHOWING WIDESPREAD INFILTRATION BY SIGNET-RING CELLS ARRANGED IN COMPACT ROUNDED MASSES.

To face page 65

The primary growth in cases of Krukenberg tumour is most commonly located in the stomach and may be so small that it escapes detection even at necropsy (Willis, 1938). In such cases the postulation of a primary Krukenberg tumour of the ovary is not generally accepted. C. J. Andrews (1947), in a review of 300 cases reported in the literature, found only three in which the ovarian tumours were believed to be primary.

Although the stomach is the commonest site for the primary growth, it may occur in other parts of the gastro-intestinal tract, Fallopian tube, uterus, pelvic connective tissue, vocal cords or adrenal glands (R. C. Costello, 1947). Owing to its small size on most occasions and the variety of places in which it may arise, it is not surprising that it may sometimes be overlooked.

Summary

A case of Krukenberg tumour of the ovary which presented as a pleural effusion and resembled Meigs' syndrome is described. The differential diagnosis, particularly in relation to tuberculous pleural effusion, with or without miliary tuberculosis, is discussed, and an attempt is made to explain the failure to demonstrate the primary growth at autopsy.

We would like to express our great indebtedness to Dr. P. Forgacs for kindly supplying us with the records of the case while at Sidcup, Dr. C. H. R. Knowles for the very complete pathological report and the photograph of the right ovary, Professor R. A. Willis for his report and helpful observations on the sections of the right ovary, and Dr. L. Woodhouse Price for the microphotographs of the sections of the lung and ovary.

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ACTIVE PULMONARY TUBERCULOSIS COMPLICATED BY ADDISON'S DISEASE

WITH A REPORT ON A CASE

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Addison's disease is a very rare complication of pulmonary tuberculosis, and indeed the case reported below was the only one that had been seen in over twenty years in the practice of a chest clinic with some 1,200 cases of pulmonary tuberculosis on its register.

The infrequent association of these two conditions was emphasised by Rowntree and Snell (1931). They quoted Minor, who had seen no case of Addison's disease during thirty-three years' work with tuberculosis, and Brown, who had seen only three cases among 2,750 deaths in patients of the Trudeau Sanatorium. Bronfinn and Guttman (1935) were interested in this problem, and believed that Addison's disease was unlikely to appear until long after any predisposing lung lesion had become quiescent or had actually healed.

Although pulmonary tuberculosis is so seldom found to precede Addison's disease, yet cases with tuberculous Addison's disease frequently have an active pulmonary lesion, as was shown by Guttman (1930). He reviewed a large series of cases of Addison's disease, among which 243 were of tuberculous etiology. There were 124 cases (51 per cent.) with tuberculous lesions of the lung or pleura, the lung lesion being active or chronic in 81 cases—a substantial proportion of the whole. Smaller series of cases have shown rather similar results, for example, Rowntree and Snell (*op. cit.*) described 57 cases of tuberculous Addison's disease. In 52 of these tuberculosis of the lungs was present, though it was active in 19 cases only. Taken in conjunction with some smaller series of cases (Conybeare and Mills, 1924; Cotton, 1935; Franks, 1950), it would seem that about one case in four with tuberculous Addison's disease may be expected to have an active pulmonary lesion.

The chest physician who is confronted with this combination and who treats the lung disease with streptomycin and para-aminosalicylic acid (P.A.S.) is bound to be interested in the effects of these drugs on the diseased suprarenals. Only one case of this kind has been recorded to date (Cheyney, 1949). The diagnosis of Addison's disease was made solely on the basis of a low blood pressure and of gastro-intestinal disturbances occurring in a patient with active pulmonary tuberculosis. Suprarenal extract was administered at the same time as streptomycin with improvement in the patient's general condition. The suprarenal extract was later discontinued without the previous symptoms recurring. The diagnosis of Addison's disease was not proven in this case and the patient's symptoms may have been due solely to the pulmonary disease. In the case described below the diagnostic criteria were stricter :

W.G. male, age 43. In 1939, at the age of 33, he first developed active tuberculosis, bilateral pleural effusions being followed by infiltration at the right apex and a positive sputum. After a period of sanatorium treatment the lesion became quiescent, and during the following year he was able to return to work.

In October 1948 he developed a productive cough, began to tire easily, and during that winter noticed unusual sensitiveness to the cold. He was beginning to lose weight. He continued to work for a further fifteen months and finally reported sick to his private practitioner in January 1951. His transfer to hospital within a few days was arranged and he came under the care of Dr. Philip Ellman.

On admission he was found to be wasted, with diffuse pigmentation of the face, trunk, arms and legs. The colour was a muddy brown and patches of slaty-grey pigment were present on the tongue, the gums and the inside of the cheeks. Some fine râles were heard in the right upper chest. His blood pressure was 90/50 mm. Hg. Other systems were normal.

Investigations.—Serum sodium 308 mg./100 ml.; plasma chloride (as NaCl) 491 mg./100 ml.; serum potassium 20 mg./100 ml. Kepler's test gave a value for "A" of 6 (normal over 20). His twenty-four-hour urinary excretion of 17-ketosteroids was 5·3 mg. His biochemical tests were considered to support the clinical diagnosis of Addison's disease. Straight X-ray of the abdomen showed no suprarenal calcification.

X-ray of the chest showed active infiltration in both upper zones with a right sub-clavicular cavity. His blood sedimentation rate (B.S.R.) was 112 mm. in one hour and his sputum contained large numbers of tubercle bacilli.

Progress.—After observation for one week he was started on intramuscular injections of streptomycin 1 G. with P.A.S. 18 G. daily. This treatment caused the slight fever that he had been running to subside. Over the next month he gained 5 lb. in weight, but showed no improvement in his Addison's disease, his blood pressure remaining unaltered and his blood chemistry showing slight deterioration. His serum sodium had now fallen to 304 mg./100 ml., and his serum potassium had risen to 23 mg./100 ml.

At this stage, in view of the failure of his Addison's disease to improve and because of the risk of sudden death in such patients, hormone replacement therapy was started. He was given daily intramuscular injections of deoxycortone 5 mg., which was combined with NaCl 12 G. by mouth. Over the next three weeks it proved necessary to increase the dose of deoxycortone progressively until he was having 15 mg. daily before an average blood pressure of 130/80 mm. Hg could be maintained. By this time his blood biochemistry had returned to normal with a serum sodium of 360 mg./100 ml. and a serum potassium of 19 mg./100 ml. After a further six weeks he was given an implant of 400 mg. of deoxycortone. A slight tendency for the blood pressure to rise was controlled by reducing the salt intake.

His streptomycin and P.A.S. had been continued without a break, and two months after admission the cavity in his right upper zone had disappeared and could not be detected by tomography. He still had some tuberculous bronchiectasis at the left apex.

By the end of five months he was fit for discharge. He had then had 145 G. of streptomycin and the disease in both lungs was restricted to small, hard areas. His sputum was free from tubercle bacilli and his B.S.R. was 30 mm. in one hour. At the time of writing, seven months after his implant of deoxycortone, no further replacement therapy has been needed.

Discussion

The case history of this patient has been recorded in some detail because it shows two apparently separate processes pursuing unrelated courses. Despite the strong presumption that the patient's Addison's disease was of tuberculous etiology, there was no evidence that streptomycin had effected any recovery in suprarenal function. The blood pressure had responded solely to an adequate dose of deoxycortone and had shown no improvement on one month's treatment on streptomycin and P.A.S., while the blood biochemistry had shown further slight deterioration. The rise in the serum potassium at this time to 23 mg./100 ml. is of some interest in view of Cayley's (1950) finding that therapeutic doses of P.A.S. may sometimes cause hypokalæmia.

There are good reasons for thinking that streptomycin or similar antibiotics are unlikely to cure Addison's disease. If tuberculosis of the suprarenals produced symptoms it might be another matter. Nolli and his co-workers (1942) were of the opinion that the asthenia found in early active cases of pulmonary tuberculosis might be due to depression of suprarenal function, but there was no indication of actual destruction of the glands. Guttman (1930) pointed out that tuberculosis of the suprarenals always seemed to be blood-borne, so that they would probably be affected early in the natural history of the patient's tuberculosis. Boyd (1944) has stated that destruction of the suprarenals is usually complete by the time Addison's disease can be diagnosed clinically, while Bronfinn and Guttman (*op. cit.*), who were studying the rare amyloid infiltration of the suprarenals, were only able to make a confident clinical diagnosis of Addison's disease in cases where amyloid infiltration was very extensive.

It would therefore appear that once clinical signs of Addison's disease have developed the suprarenals are too badly damaged for streptomycin to help. The correct use for this drug must surely lie in its prophylactic effect in healing the hidden lesions in the suprarenals while it is being used to treat open disease of the lungs or in other systems. We shall only learn of such cases by the later appearance in X-rays of the abdomen of tell-tale spots of calcium in the suprarenal areas, mute witnesses to the cases of Addison's disease that were never allowed to develop.

Summary

A case is described of Addison's disease in conjunction with active pulmonary tuberculosis. Streptomycin and P.A.S. were used with benefit to the lung lesion, but without apparent effect on the Addison's disease. The latter was satisfactorily controlled by deoxycortone.

The development of tuberculosis of the suprarenals is discussed, and it is suggested that streptomycin is likely to be valuable as a prophylactic measure rather than as a means of treating established Addison's disease.

I should like to thank Dr. Philip Ellman for permission to publish this case and for help in the preparation of this paper.

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REVIEWS OF BOOKS

Die Tuberkulose: Ihre Erkennung und Behandlung. Edited by Prof. Dr. HELLMUTH DEIST and Prof. Dr. HERMANN KRAUSS. Stuttgart: Ferdinand Enke Verlag. 1951. Pp. 754. 297 illustrations.

In this substantial volume the authors have made it their main object to stress the teaching of Sauerbruch and Schroeder, who considered tuberculosis as a general infection and not as a disease confined to one organ. That is why the authors felt justified in presenting the whole subject of tuberculosis in one book, which is divided into thirteen chapters. Each section is written by a different author with special experience in his respective field of tuberculosis. The book is generally well presented and the radiographs, diagrams and photographs of pathological specimens are excellent. The quality of each chapter, as far as one observer can judge, is of a high standard and contains a remarkable amount of information. There is a valuable survey of the general pathology, bacteriology and immunology of tuberculosis. Diagnosis and differential diagnosis of pulmonary tuberculosis are ably discussed from the clinical and radiological point of view. There are a few minor errors—*e.g.*, the differential diagnosis between apical tuberculosis and Pancoast tumour. A Pancoast tumour is not only characterised radiologically by erosion of ribs, since this may occur in any bronchial neoplasm, reaching the chest wall. More important are the location in the superior pulmonary sulcus and the involvement of the cervical sympathetic, often producing Horner's syndrome. Illustration No. 72 shows a bronchial carcinoma in the left upper mediastinum and not a Pancoast tumour as referred to in the text. The chapter on tuberculosis of bones and joints gives a brief but up-to-date survey of the subject and an admirable discussion on the radiological differential diagnosis of tuberculosis of the spine. The sections on tuberculosis of childhood, tuberculosis of the skin and the urogenital tract are well presented. There are also interesting chapters on tuberculosis of the eye and ear. The gastro-intestinal tract has been somewhat neglected and is mainly described from the surgical point of view. The surgical treatment of pulmonary tuberculosis is thoroughly discussed and illustrated with particularly well-designed diagrams. General and medical treatment of tuberculosis as well as the modern chemotherapy are extensively discussed. Special mention is made of Conteben, a preparation introduced by Domagk, which arrests the growth of tubercle bacilli in the test-tube in a concentration of 1 : 1,000,000. Striking results with this preparation are claimed in the treatment of ulcerative lesions in the upper air passages.

A major defect of the book is the absence of a bibliography at the end of each chapter. More references to English and American contributions would have been desirable, but this omission may be explained by the long isolation of Germany.

Although this work cannot replace textbooks on the various subjects of tuberculosis, it is a valuable addition to the literature, since the authors succeed in presenting the whole problem of tuberculosis in a concise and clearly written form. The book is a useful and informative volume for the library of physicians and surgeons interested in tuberculosis.

A. E.

The Electrocardiogram in Pulmonary Tuberculosis. By ALLAN BJÖRKMAN. Stockholm, 1951.

The author of this painstaking monograph produces convincing evidence, based mainly on statistics, that pulmonary tuberculosis tends to produce flattening of the T waves—and to a lesser extent of the P waves—in the classical (bipolar) limb leads; but the chest leads receive practically no consideration.

He shows that this occurs to a greater extent than can be accounted for by such factors as change in position (or rotation) of the heart, accelerated pulse rate, or the effects of autonomic instability. It is paralleled by the similar T wave changes which often accompany rheumatic fever, and he therefore considers that the likely reason for these flattened T waves is myocardial damage (perhaps anaphylactic) caused by the tuberculous toxins, rather than any mechanical effects on the heart produced by the lung lesions (*e.g.*, increased pressure in the lesser circulation). Moreover, as he reminds us, myocardial lesions, such as interstitial myocarditis, Aschoff's nodules or brown atrophy, have quite often been reported at autopsy in cases of pulmonary tuberculosis. But he was unable to find—in an admittedly limited series of cases—any correlation between the degree of T wave flattening and the severity of the disease, as judged by the radiographic appearances, amount of fever and/or raised sedimentation rate.

Perhaps the most valuable feature of the monograph is the detailed review of the relevant literature, which has also been well tabulated. The translation (by K. M. Lindsborg) is very adequate, and the English clearly expressed though not always entirely immaculate. There are a few misprints. The monograph more than adequately covers its limited subject, and every aspect seems to have been considered—except possibly the influence of endocrine factors, which is almost entirely omitted. Nevertheless, in your reviewer's opinion, Dr. Björkman's conclusions are correct, for flattened or deformed T waves are a well-known finding in many types of toxic myocarditis.

D. R.

Chest X-ray Diagnosis, by MAX RITVO. Henry Kimpton. 1951. Pp. 558 + 615 illustrations. Price 105s.

In this sumptuous volume of over 500 pages, covering most thoracic and intrathoracic and upper respiratory diseases, only eighteen pages are devoted to pulmonary tuberculosis. This cursoriness hardly reflects the relative importance of the condition as a cause of (intrathoracic) disease, and the whole section is somewhat ill-ordered and vague. In spite of the apparent need for economy of words and illustrations, miliary tuberculosis is twice referred to, once under childhood tuberculosis and again, only a few pages farther on, in a section on adult tuberculosis. The two parts might conveniently have been joined together.

The difficulty of demonstrating the primary lesion is glossed over, and only cases showing a relatively massive shadow are mentioned. No tomograms are shown, and the value of this method in the demonstration or exclusion of pulmonary cavities is not mentioned. Nor is a lateral view given in any of the cases revealing an opacity near the hilum, to show whether the shadow is possibly the result of glandular enlargement or of a lesion lying in the lung behind the hilum.

There are interesting sections on the pneumonias and on histoplasmosis which might concern the phthisiologist, though there is a tendency for the clinical matter to be more fully described than the radiological appearances.

On the whole the illustrations show what is intended, though somewhat larger localised views might have helped to show some of the smaller tuberculous lesions more clearly.

G. S.

Approach to Cardiology. By J. CRIGHTON BRAMWELL. Oxford University Press. 1951. Pp. 197. Price 17s. 6d.

The pressing need for medical education to establish a closer liaison between the pre-clinical and clinical subjects and to bridge the gulf between them has had no greater exponent than that distinguished physician and cardiologist Professor Crighton Bramwell. There is no doubt that students benefit when, passing from the pre-clinical to the clinical period, they can profit from a system wherein members of the clinical staff are also attached to departments such as those of anatomy and physiology. In a study of closely related conditions like diseases of the heart and lungs the applied knowledge of, for example, cardiodynamics, the anatomy of the bronchopulmonary segments and respiratory physiology is of inestimable practical advantage to the student new to the wards.

In his monograph on the " Approach to Cardiology " Professor Bramwell illustrates in a practical manner some fundamental general principles of cardiology in relation to the pre-clinical sciences, although this approach involves no less than the whole field of clinical medicine.

The clinical approach, comprehending a study of the patient as a whole, encourages a proper outlook on a group of disorders ranging from slight abnormalities of function to mortal disease. Professor Bramwell emphasises the need for an accurate history which will reveal not only the clinical features but also the social, psychiatric, occupational and other factors.

It is not surprising that so experienced an examiner and teacher should express concern at the " danger of students being so overburdened with factual knowledge that they can no longer see the wood for the trees." His approach is one that will enable students to think out clinical problems for themselves against a clear-cut anatomico-physiological background. As Professor A. V. Hill, who collaborated with him in Manchester, acknowledges in his foreword, " I learnt a little of the principles on which methods and ideas of physiology can be employed in clinical medicine."

In an excllent chapter on the etiology of heart disease Professor Bramwell refers to " secondary heart disease " and the close partnership between diseases of the heart and lungs, as evidenced by the chronic pulmonary disorders such as emphysema which are known to produce ultimately a cor pulmonale.

The style of the book is lucid and pleasing. We can recommend Professor Bramwell's treatment of his subject and his selection of illustrations of cases for enjoyable reading and for a sound approach to clinical problems in general.

P. E.

Chronic Bronchitis. By TREVOR HOWELL. Butterworth and Co. Price 17s. 6d.

It is very difficult to decide for whom this book has been written. In his preface the author states that his experience is derived from general practice, the Royal Hospital, Chelsea, the Army in India and a geriatric research unit at St. John's Hospital, Battersea, and hopes that his account will " not be entirely lacking in interest." It is by no means lacking in interest, but is marred almost throughout by the drawing of unwarranted statistical conclusions from small uncontrolled series of patients; further, it is not always clear

in the text whether the patients referred to are Chelsea Pensioners or civilians. The book would be far more readable if the author had completed his questionnaire upon his fifty-three patients and then merely used his figures as a basis for generalisations. In the chapter upon Clinical Pathology the figures are frankly confusing. In a table showing the relative frequency of organisms in the sputum, *B. Friedländer* was found in 12 per cent., a startling enough figure in itself; but as the total number of organisms found adds up to only 90 per cent., one is left wondering whether the remaining 10 per cent. of patients had sterile sputa.

The chapter upon morbid anatomy deals with the autopsy findings in 300 bronchitics. Many of the data are of considerable interest, but, listing the causes of death, no mention is made of emphysema, which was found in greater or lesser degree in 100 per cent. of cases. " Chronic bronchitis alone " is listed as the commonest cause after " right ventricular failure "; an explanation of the mechanism of death in such cases would be an advantage.

Dr. Trevor Howell has been at pains to cover relevant medical literature, and the historical introduction is well documented. Also, his remarks upon treatment are carefully thought out. His book may be read with profit, but one is left with the uneasy feeling that many of his statements require considerable substantiation before they can be accepted. N. C. O.

Phtisiologues et Phtisiologie. By ETIENNE BERNARD, Professeur à la Faculté de Médecine de Paris. Masson et Cie, Editeurs, Libraires de l'Academie de Médecine, 120, Boulevard Saint-Germain, Paris (VI[e]). 1951. Price 1,200 francs.

This is a book with a difference. The author claims that he has yielded to his inclination to express his ideas in a vivid and vital manner, and he has attempted this by throwing into relief part of the life story of the men who have contributed to our knowledge of chest diseases. The sketch of Pierre Ameuille and his contributions to our knowledge of the bronchi in health and in disease is followed by Fernand Bezançon and his work in correlating many of the clinical and laboratory features of tuberculosis. The melancholy story of Michel Leon-Kindberg and the particularly sad end of his life is set forth in a short appreciation of his work. Pneumothorax treatment is coupled, as one would expect, with the name of Forlanini. The discussion is not very detailed, but the author prefers to persist with pneumothorax treatment for five years on the average, and he criticises " the Anglo-Saxons," who, he states, appear to think that three years is sufficient.

A discussion on streptomycin follows, and this is coupled with the name of S. A. Waksman.

The remainder of the book contains a series of essays on the campaign against tuberculosis in Belgium, an account of the September 1950 meeting in Copenhagen, and a somewhat detailed discussion of the World Health Organisation and its relation to measures of tuberculosis prevention.

J. M.

BOOKS RECEIVED

An Atlas of General Affections of the Skeleton. By H. A. T. Fairbank. 55s.

Chest Surgery for Nurses. By J. Leigh Collis and L. E. Mabbitt. Pp. viii+188, with 117 figures. Third Edition. 12s. 6d.

Food and Nutrition. By E. W. H. Cruickshank. Pp. 443, with 51 illustrations. Second Edition. 30s.

The Care of the Ageing and Chronic Sick. By A. P. Thomson, C. R. Lowe and T. McKeown. Pp. 133. 17s. 6d.

Thoracoplasty in the Treatment of Cavernous Tuberculosis of the Lung. By Erling Refsum. Acta Tuberculosia Scandinavica. Supplement XXIX. 1951.

Pathological Histology. By R. F. Ogilvie. Pp. 506, with 295 coloured photomicrographs. E. and S. Livingstone, Ltd. 40s.

Untersuchungen über das Weisse Blutbild und das Sternalpunktat bei Primärem Lungenkarzinom. By Jorma Pätiälä. Acta Tuberculosia Scandinavica. Supplement XXVIII. Helsinki, 1951.

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REPORTS

EFFECT OF WELDING ON HEALTH: A RECENT INVESTIGATION

An investigation into the possibility of ill-health arising from the welding process has been carried out by the Factory Department of the Ministry of Labour and National Service, and the results have been published in a book entitled " The Health of Welders."*

The investigation consisted of a survey of the various types of welding followed by clinical examination of some 250 welders in different industries, supplemented in many cases by special examinations such as radiological examination of the chest and blood examinations. Literature on the subject, particularly that published during the past fifteen years, has also been reviewed.

The main conclusions are that welders do not suffer from any specific disease that could be described as " welders' disease," nor does occupational dermatitis appear to be a frequent or serious cause of disability. Electric welders may suffer from " arc eyes," but this has no permanent effect on the vision. Electric welders also suffer to a greater extent than other workers from a slight superficial inflammation of the eyelids.

Amongst welders exposed to high concentrations of fumes slight irritation of the throat is not uncommon, but no serious effects on the throat or nose were observed. Symptoms, indicating a mild form of bronchial irritation, were higher than would be expected in a comparable non-welding group. Exposure to welding fumes does not, however, predispose to pulmonary tuberculosis. Stomach troubles are no higher in welders than in the general population, rheumatism is not excessive, there is no evidence of ill-effects on blood pressure or the nervous system, and where there is good or moderately good ventilation there is little danger of gassing during welding.

Some general recommendations are made, but it is pointed out that as the main risk of ill-health amongst welders is due to fumes, the remedy is ventilation. The difficulty, however, is to decide when and where special provision should be made, and as there is such a wide variation in the different welding processes it is considered that the problems of each firm will have to be looked into individually. The general recommendations include:

1. No special precautions are needed where only occasional welding jobs are done, unless considerable quantities of very toxic elements are present.
2. Where oxy-acetylene welders are working there should be the same good general ventilation as for workrooms where fairly hot processes are carried out.
3. Close localised exhaust ventilation should be applied where articles are electrically welded on benches or stands.
4. In the welding of large articles of mild steel, such as vehicles or prefabricated parts of bridges, general ventilation should be relied upon if good practical methods of localised exhaust cannot be evolved.
5. Exhaust ventilation should be provided where the fumes contain substances in sufficient quantity to be poisonous or irritating.
6. Where welding is done in confined spaces exhaust draught should be provided as close to the welding point as possible.

* " The Health of Welders," by A. T. Doig, M.D., D.P.H., H.M. Medical Inspector of Factories, and L. N. Duguid, B.Sc., A.M.I.(Mech.)E., M.I.W. Obtainable from H.M. Stationery Office or through any bookseller, price 3s. net, post free.

The St. Thomas's Hospital Reports. Second Series, Vol VI.

THIS volume contains some of the work completed in the hospital during the year 1950.

The articles cover a wide range of topics, and many of them are highly specialised. However, the two on diabetes are of some general value, whilst there are three others whose content will be of more particular interest to readers of this journal.

The first of these reviews thirty-one cases of Addison's disease. It agrees with the more recent series in showing that tuberculosis is giving way to simple atrophy of the suprarenals as the major cause of the condition, and after stressing the incidence of mental symptoms rather more than usual, suggests that the prognosis—although improved by endocrine therapy—is still by no means good. As a means of improving this, the suggestion is made that streptomycin and P.A.S. might be exhibited in tuberculous cases, and more careful endocrine supervision given to all.

The second of these articles reviews 490 cases of carcinoma of the bronchus. This covers every aspect of the disease and states yet again the gloomy prospects of sufferers from this disease, and exhorts one to consider more often the presence of carcinoma in patients complaining of chest symptoms.

The last of these three articles presents case summaries of apical pneumonia in childhood, and in view of the frequent incidence of upper respiratory infection prior to the pneumonia, suggests that inhalation may be of importance in the pathogenesis.

J. E.

Brompton Hospital Reports. Vol. XIX. 1950.

THIS volume, edited by Drs. Maurice Davidson and A. F. Foster Carter, consists of articles by members of the Brompton Hospital staff which have, with two exceptions, already been published in medical journals during 1949.

Of the two papers not previously published, one, a paper on " A Ten-year Follow-up of Patients with Tuberculous Empyema complicating Artificial Pneumothorax Therapy," is a piece of original research on Brompton Hospital records undertaken by Dr. J. R. Bignall, the Senior Medical Assistant in the Institute of Diseases of the Chest, and is presented as a report to the Medical Committee. It is a subject of current interest and is of considerable practical significance. The other article is by Dr. Foster Carter, on the " Education and Management of the Sanatorium Patient," and is based on a lecture given at the Institute of Diseases of the Chest. It deals with sanatorium routine and discipline, occupational therapy, vocational guidance, etc., and is full of sound, balanced advice based on experience.

The papers already published comprise a wide variety of subjects, including " The Centenary of the Brompton "; the Harveian Oration for 1949 on " Individuality in Medicine," by Dr. Geoffrey Marshall; " The Management of Pulmonary Tuberculosis," by Dr. F. H. Young; " The Management of Acute Pneumonias in Adults " and " Sarcoidosis," by Dr. Scadding; " The Early Diagnosis of Bronchial Carcinoma," by Mr. Brock; " Pulmonary Changes in the Reticuloses," by Dr. Oswald; " Pulmonary Disease in the Elderly," by Dr. Livingstone; and " Favourite Prescriptions in Pulmonary Diseases," by Dr. Lloyd. There are also two interesting papers on " The Radiology of the Heart and Lungs," by Dr. Simon. This volume certainly maintains the high standard that we have come to expect and is, as usual, very well produced.

N.A.P.T.

Annual Report of the National Association for the Prevention of Tuberculosis, 1950-1951. "Hope in Tuberculosis."

THIS report brings with it the message of "Hope in Tuberculosis," both from the preventive and therapeutic aspects, and in spite of difficulties of which the Association is fully aware.

Until very recently there were in Great Britain more than 400 deaths each week, but more recently the figure has dropped to nearer 300. Although it is recognised that tuberculosis is only a part of the National Health Service's manifold activities, the report expresses the hope that control of this disease will be made even more possible for every man, woman and child, for today facilities near his own home, at the cost of the service and with the attention of a specialist during the whole course of the illness, are readily available.

The report recognises the many problems assailing the anti-tuberculosis campaign from all sides. For example, the facilities for sanatorium treatment are often less favourable than a few years ago—a patient has, on the average, to wait some six to nine months for a sanatorium bed. The Tuberculosis Service is short of some 4,500 beds, mainly owing to the lack of nurses and domestic personnel, and in this connection the recommendations of the Association with regard to the "secondment" of student nurses deserve attention. It is essential that the waiting list of something like 10,000 should be reduced.

The welfare of the tuberculous patient and his family forms the background to medical treatment, and less has been achieved in this mainly preventive sphere than on the curative side, where, the report acknowledges, there is cause for a modest satisfaction. Once again integration of the preventive and therapeutic services is urged.

The report includes in its survey such problems as social welfare, Commonwealth Associations, the part played by local health authorities, home treatment, rehabilitation, research, treatment in Switzerland, the Tuberculosis Educational Institute, and other of the wide activities of the National Association for the Prevention of Tuberculosis which have been noted in this journal during the course of the year.

N.A.P.T.

"HOME WORK" FOR TUBERCULOUS PATIENTS

THE National Association for the Prevention of Tuberculosis has recently published "Home Work with a Difference," a survey by Muriel Owen-Davies, N.A.P.T. Research Scholar.

The survey was carried out in Surrey over a period of eighteen months, and the report shows the value of remunerative employment for tuberculous patients confined to home. Such work can give the patient many benefits—an interest in life, a feeling of being useful in the community, besides some money in his pocket. Local authorities are responsible for looking after tuberculous patients and have powers in this direction, but so far no home work schemes have been established. One of the most valuable parts of the report is the frank statement of the difficulties involved in arranging home work—*i.e.*, obtaining the work, getting it carried out to time and to a high standard, disinfection, and dealing with financial complications with regard to capital expenditure, the reduction of allowances, etc. The case papers

attached to the report as an appendix are very full and most interesting as practical examples not only of difficulties involved, but of successes achieved and of possible developments.

The Research Scholar's conclusion is that an organised home-work scheme set up by a local authority is practicable and would be beneficial, but that it would have to be subsidised, probably continuously, though in course of time on a smaller scale. It is obvious that the existing facilities for sheltered employment for these tuberculous patients in need of it are very limited. The present number of such workshops is inadequate to the need, and well-organised home employment facilities, as outlined in this survey, should certainly be available in every county.

CANADIAN TUBERCULOSIS ASSOCIATION

Death Rate drops to Record Low

A further significant decline in Canada's death rate from tuberculosis is reported in the preliminary figures for 1950 just released by the Dominion Bureau of Statistics. The new figures of 25·9 per 100,000 is a record low and represents a total of 3,582 deaths across the Dominion. These figures are noteworthy in that they include, for the first time, the new province of Newfoundland.

It is of note that the overall rate for Canada has fallen, for the first time in our history, to the twenties. The 1949 figure of 30·4, the previous all-time low, was still hovering above the line. Now we have reached the same decade as the United States, which for 1950 reports a provisional death rate of 22·2 per 100,000.

The honour of having the lowest rate in the Dominion goes again in 1950 to Ontario with the exceptionally low figure of 13·0. This a record of which to be justly proud. Saskatchewan came next with a rate of 18·5, and the other provinces in order are: Alberta, 19·3; Manitoba, 22·8; Nova Scotia, 26·1; British Columbia, 27·6; Prince Edward Island, 30·2; New Brunswick, 30·5; Quebec, 39·3; and Newfoundland, 68·5.

Report of the Co-ordinating Committee on Abstracting and Indexing in the Medical and Biological Sciences. Published by the United Nations Educational, Scientific and Cultural Organisation in Paris, 1951.

This informative pamphlet summarises the decisions made in regard to medical abstracts by the Unesco Conference up to the end of 1946. It describes how after the war it was important to renew contacts and the exchange of ideas between one nation and another, but it was also felt that this should be done at lower cost and with less overlapping than in pre-war years. The Unesco Secretariat undertook to co-ordinate the abstracting of scientific literature, and their findings at several conferences are here reported.

Some idea of the scope of the work may be had when it is appreciated that between one and two million articles appear annually in fifty thousand separate periodicals, and the classification of the abstracts, according to both time and subject, is a tremendous undertaking. It is essential that there should be some kind of uniformity, particularly as regards references, abbreviations and degree of summarising, and it is this that the Secretariat has set out to achieve. Time alone will show how far it is successful, but any scheme to produce harmony and coherence in so international a field as science is to be welcomed, and the thoroughness of the report betokens a satisfactory outcome.

World Health Organisation. WHO/TBC/43. 27th October, 1951. Tuberculosis Control Programme in Finland.

THIS report, published in October 1951, covers an investigation made by Dr. Erkki Larmola between May and August of that year. It deals comprehensively with such factors as population, mortality, morbidity, mass radiography and B.C.G. vaccination. Under the heading of environmental factors Dr. Larmola discusses climate, occupation and economic questions as well as the state of housing and nutrition and the social habits of the people. He includes also in his survey the organisation of the Finnish Tuberculosis Services, care and rehabilitation, and the position of under- and post-graduate teaching in his country.

With regard to control of the disease in the near future, he proposes the following tasks:

1. To intensify the network of the tuberculosis dispensaries all over the country up to the point where one dispensary area will comprise at the maximum 70,000 inhabitants, and to ensure good communications to the dispensary from all communities of the district.
2. To develop mass radiology so extensively that the threatened groups of population really will be under effective control and the new cases diagnosed in their early phase.
3. To expand and develop the central sanatoria of the tuberculosis districts in order to shorten waiting time, lengthen the average period of treatment and enable the sanatoria to accomplish, in addition to their clinical duties, social care and rehabilitation work.
4. To plan and develop a general rehabilitation programme for those in need of it and an after-care programme for the "good chronics."
5. To train sufficient workers for tuberculosis control as to remedy the present extremely serious scarcity of personnel.

He concludes his report with an analysis and evaluation of the tuberculosis problems in Finland: Tuberculosis control is relatively young in Finland; it has been done methodically only for a few decades, on a large scale and with the aid of public money only for a little over twenty years. It must also be remembered that the same period is marked by the trail and difficulties of the war, which have badly handicapped all social work and constructive effort. All resources, both human and financial, of the nation have been needed for a great number of other necessary and indispensable things. It is thus comprehensible that the tuberculosis situation is still worse in Finland than in most other civilised countries.

In spite of all difficulties the anti-tuberculosis work has been carried on with purposeful energy and industry. The results achieved are considerable. It has been possible to profit by the experiences of other countries, notably of the Scandinavian neighbours, but at the same time methods and forms of our own have been evolved, suited to the conditions and economic possibilities of the country. Results are visible: tuberculosis is rapidly diminishing. It is probable that Finland will soon reach the status of the other civilised countries and then be able to concentrate work not only on reducing the losses caused by tuberculosis but on complete eradication of this disease.

NOTES AND NOTICES

NAPT LUNCHEON TO SIR ROBERT YOUNG

THE members of the Council of the National Association for the Prevention of Tuberculosis gave a private luncheon on Thursday, November 15, at the Connaught Rooms to Sir Robert A. Young, the vice-chairman, in celebration of his eightieth birthday. The Duchess of Portland presided at the luncheon, and, on behalf of the Council, presented Sir Robert with a gift of books.

NAPT

THE third Commonwealth and Empire Health and Tuberculosis Conference will be held in London at the Central Hall in July 1952.

The Lectures and Discussions will take place on July 8, 9 and 10, and the morning of July 11.

There will be visits to Sanatoria, Hospitals and Clinics on July 11, 12 and 13.

THE THORACIC SOCIETY

THE following arrangements have been made for this year's meetings:

Spring Meeting: February 22 and 23, 1952. Royal College of Surgeons, London.

Summer Meeting: July, 1952. Sully, Glamorganshire.

NATIONAL TUBERCULOSIS ASSOCIATION OF AMERICA

THE annual meetings of the National Tuberculosis Association and its medical section, the American Trudeau Society, will be held in Boston, Mass., from May 26 to 29, 1952. The National Conference of Tuberculosis Workers will meet at the same time.

Invitations are being extended to chest specialists and investigators in the tuberculosis field to submit abstracts of papers for presentation at the meeting. The scientific sessions will be planned early in 1952 after the abstracts have been reviewed by the Medical Sessions Committee of the Programme Committee.

In addition to seven sessions at which scientific papers will be presented, there will be six panel discussions on subjects of immediate medical interest. Subjects tentatively scheduled for the panels are: chemotherapy in tuberculosis, bacteriology, ACTH and cortisone, excision surgery, pulmonary suppuration, and mycobacterial diseases other than tuberculosis. There will also be a number of seminars for small groups.

Dr. Herbert L. Mantz of Kansas City, Mo., is general chairman of the Programme Committee; Dr. Theodore L. Badger of Boston is chairman of the Medical Sessions Committee; and Dr. Lloyd Florio of Denver is chairman of the Programme Development Sessions Committee.

HONOURS

THE King has conferred knighthoods upon Dr. Geoffrey Marshall and Mr. C. Price Thomas and invested them with the insignia of Knights Commanders of the Royal Victorian Order.

Our readers will wish heartily to congratulate them on this well-merited distinction.